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## MUMU

COMMANDER R. N. BABIONE, M.C., U.S.N.

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J. E. STRODE, M.D.

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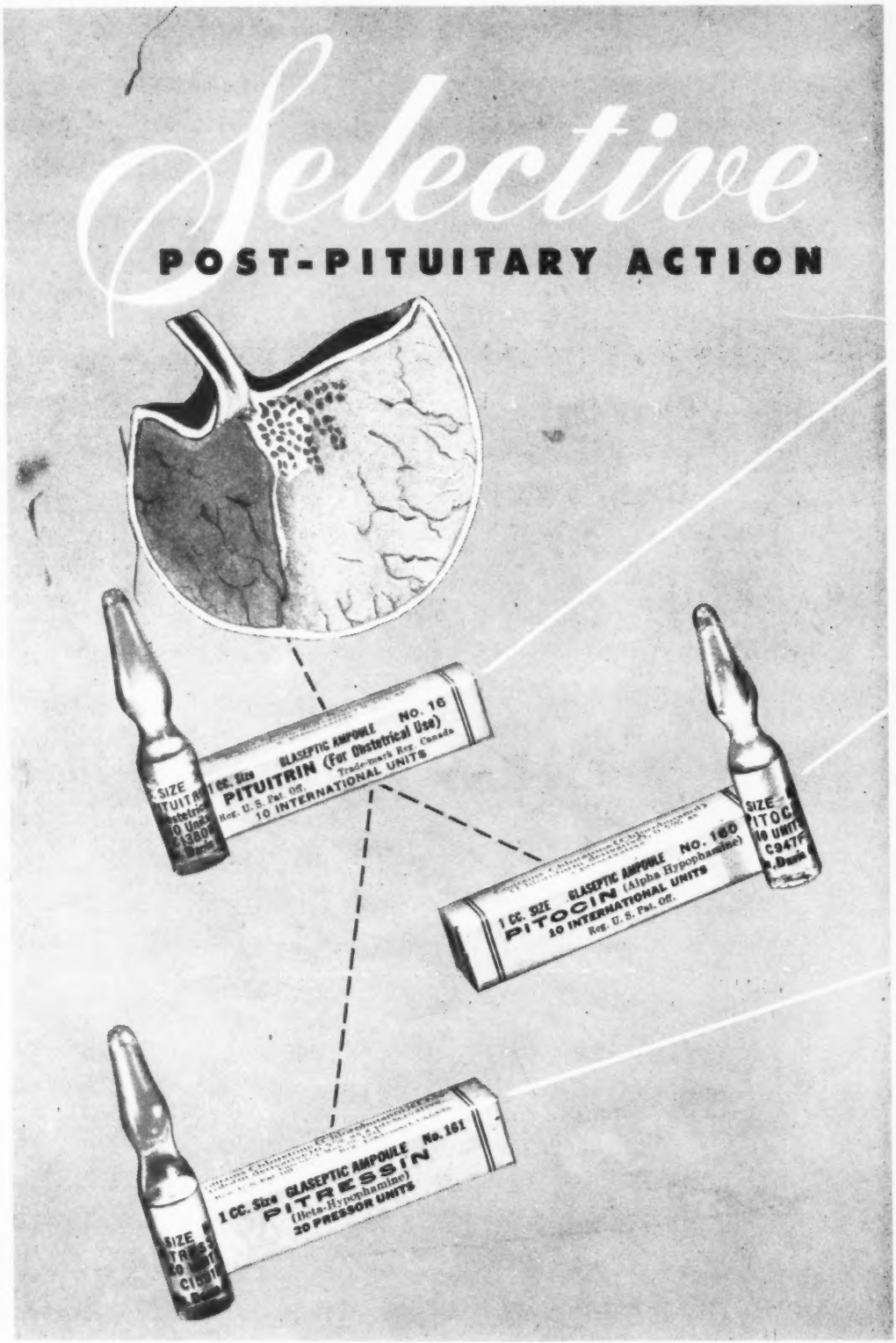
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# Mumu

## A Few Facts About Filariasis for Folks Who Fear That Filaria-infected Fellows Will Fetch Filariae from the Front

Commander R. N. Babione, M.C., U.S.N.

Let's get acquainted with *mumu*, the Samoan word for "red." This word is about to become popular in medical literature as the best fitting handle for the allergic, or early, phase of filariasis[1]. This is the only phase of the disease so far seen in the men who recently contracted filariasis in the Samoan and other areas. It bears the same relation to elephantiasis that hay fever bears to nasal polyps. The one is a temporary edema, which will subside when the allergen is removed; the other is a loose connective tissue proliferation, and it does not subside. Elephantiasis is a terminal stage following tens or scores of years of repeated insults to the lymphatic system, usually involving pyogenic bacteria as well as worms. It does not necessarily follow worm infestation, and is not expected as a sequel to simple *mumu*.

Let us confine our attention to the less well known early phase of filarial infestation. The victims call it *mumu*, which is the term the Samoans gave it. This is fitting because, in addition to the swelling, there is often a red streak or patch. This is broader than a true lymphangitis, and, unlike the latter, frequently spreads down the arm or leg instead of towards the body. A very similar swelling and redness is sometimes noted in sensitized persons around the site of a typhoid injection.

These swellings come and go, frequently are brought on by exercise, and disappear fastest during rest. They sometimes seem to migrate, appearing at the shoulder one week, the elbow the next week, the forearm later, and perhaps at the wrist finally. Sometimes, after the swellings and migrations cease, a knot or thickened lymphatic channel remains at the site of the last swelling. If this is dissected out, and sectioned carefully, a dead adult filarial worm will often be found*. If the worm has been dead for some time, it will show as a degenerated or calcified mass, with a foreign body reaction around it. Living, motile, adult worms have been dissected free on at least two occasions[2]. They have also migrated out of sections of freshly excised nodes, placed in saline[3].

The manifestations are characteristic of allergy. There is often a marked eosinophilia of the blood. Forty per cent is not rare. The peri-lymphatic tissue around a worm is filled with eosinophils. When an extract of another filarial worm (Dirofilaria immitis) is injected intra-cutaneously, a swelling and redness occurs which is exactly like *mumu* itself. Unexposed persons usually do not show this: their skin test is negative. The skin tests of persons bitten by filaria-infected mosquitoes become positive *before* clinical symptoms of *mumu* appear[2]. This may occur in a few months if the victim receives many bites and if he is a type of person easily sensitized. In one small series, 7 per cent of young adult males reacted positively before exposure[2]. This finding may be the result of prior sensitization to other round-worm antigens, *e.g.*, Trichinella spiralis. It may have been such persons who showed the abnormally short incubation periods of three months, from exposure to first symptoms of *mumu*.

Diagnosticians should remember that *mumu* patients rarely if ever show micro-filariae, or baby worms, in the blood. Conversely, those persons whose blood is teeming with micro-filariae are not bothered with any symptoms of *mumu*. This is a little known concept: Either the worms and the man bother one another, in which case the man has *mumu*, and the worm becomes surrounded by edema and eosinophils every time she extrudes a foreign protein: *or* the man and his worms get along well together, the man free of symptoms and the worms busy procreating unimpeded[4]. The latter stage may occur in white people who become "desensitized" by large and repeated doses of worm protein. In fact, a bad attack of *mumu* may render the skin test temporarily negative (cf. the anergic phase of the tuberculin test).

The timorous citizen and the alarmed Public Health Officer need not fear the victims of *mumu*, but rather the perfectly asymptomatic natives of Samoa or Puerto Rico. Their dread of the poor *mumu* case will evaporate when they realize how much exposure is required in order to develop a good producer of micro-filariae. Even in Samoa, where transmission is intensive, micro-filariae are not often at demonstrable levels in the blood of children under ten years of age, and have been found only once in that of a child under three years of age. Visualize babies and young children, with absolutely no protection from mosquito bites, living in close contact for years with parents whose blood is teeming with micro-filariae, and try to estimate the number of worms they have received before becoming a demonstrable source of filariasis. It is doubtful that service men from Samoa have had even a small fraction of that amount of exposure.

*The adult worm is about as thick as a coarse hair, and 1½ to 3 inches long. It is usually coiled.

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This leads us to believe that few of these *mumu* cases will ever show micro-filariae in the blood, and those that do finally reach that stage will show scant numbers compared to the average "native." So far, at least, no micro-filariae have ever been found in the blood of cases among the troops.

To account for this fact, a hypothesis has been based upon the miles of lymphatics in which the worms live. Male and female worms must mate before baby worms can be produced. Perhaps it would take scores or even hundreds of worms in the body before a single mating would be likely to occur. On further thought, recalling that these worms possess motility, the lymph nodes would seem to afford a convenient trysting place, and even in worms it should be presumed that love will find a way.

Furthermore, against this hypothesis of non-mating, stands the fact that gravid female worms have been reported in tissue sections, and found alive in lymph nodes, in cases of *mumu* which showed no micro-filariae in the blood[3]. There are probably several reasons for the failure to find microfilariae. First, we do not know how thoroughly the swelling of *mumu* interferes with the free migration of micro-filariae through the lymphatics. Secondly, we do not know how many reproducing female worms are needed to stock the blood with a discoverable number of microfilariae in each cubic centimeter of blood. Lastly, we suspect that micro-filariae do not circulate long in sensitized persons. It has been reported that micro-filariae have been found in the blood of the recipient as long as six weeks after a transfusion with blood rich in micro-filariae. (They do no harm, cannot multiply, and eventually die.) The recipient was probably not sensitized to worm protein at the time of the transfusion. But we may offer a hypothesis that in cases of *mumu,* which are highly sensitized to worm protein, the baby worms are picked off rather quickly after their release, thus preventing accumulation.

The fact that so far no micro-filariae have been found in the cases of the present war is no assurance that they will not someday appear. But we have seen that it probably takes many worms to sensitize a man, and many more to desensitize him. The stage of *mumu* means that the first worm assault is in progress and is being resisted. At this point we now remove the case from exposure to more worms. If the worms are not too numerous, they will all die before the man becomes desensitized. We know that many worms do die. It is possible that some of our troops received such heavy infestations in a short time that, in spite of worm casualties, some worms may survive until the man becomes desensitized. If enough worms should survive to this point, they might be able to keep the man desensitized, and go into production. In view of the findings in native children, such cases would be a rare exception. And even if they should produce some baby worms, they could never approach the production rate found in the Samoan adults who have had 20 to 100 times this much exposure to filarious mosquito bites. Furthermore, in their communities, they will be lone producers, not aided by 20 per cent of the remainder of the population as was the case in the villages in which they contracted *mumu*.

In any event, people enjoying American standards of living would not tolerate the number of mosquito bites which produced our Samoan cases of *mumu*. Remember, too, that our "menace" must go on producing micro-filariae year after year for many years in order to produce secondary micro-filaria-carriers. This he will be unable to do unless he reinfects himself. His original stock of worms will die of old age before he could produce a secondary distributor of micro-filariae.

Filariae don't fare well in the United States. There was once a pool of filariasis around Charleston, S. C. It has practically died out. Many laborers from the West Indies have been imported to East Coast States with their blood teeming with microfilariae. No alarm has been noted. Our troops with *mumu* have no demonstrable micro-filariae, and probably most of them never will have. Certainly they are far less dangerous than these West Indian laborers.

It may be further reassuring that, unlike malaria organisms, worms do not multiply in the mosquito. Instead, they undergo a necessary moult or two, in order to become a penetrating worm, then crawl into the mosquito's proboscis. They come out of the mosquito, one worm for each worm that went in. The mosquito doesn't like it either. It probably makes her tired, having those worms shedding their skins in her thoracic muscles. At any rate, filaria-infected mosquitoes appear to fly very short distances, rarely over 25 yards from native habitations. From this fact comes our best method of prevention, to keep troops out of native villages when mosquitoes are biting. And keep the natives out of military encampments.

Lastly, filaricidal drugs are known, and their use will be perfected. At present, they aggravate symptoms when given to *mumu* cases in effective doses[5]. This finding was predicted by those who believed that allergy was the cause of symptoms of *mumu*. Dead worms give off a protein at a much more rapid rate than live ones. An alarming reaction also follows administration of filaricidal drugs in dogs which are heavily infested with D. immitis, the dog heart worm[6].

We have seen that we need not be worried any longer over *mumu* cases. Most of them will never show micro-filariae in the blood. Those very heavily infested patients who pass through the phase of *mumu* to the stage of desensitization with surviving worms, are likely to show few if any micro-filariae in the blood until desensitization occurs. Once the patient is desensitized, mass slaughter of adult worms should be possible without aggravation or exacerbation of symptoms.

## SUMMARY

1. Our cases suffer *mumu,* an allergic response to worm protein, but do not produce micro-filariae *in the blood,* at least in demonstrable numbers.

2. It takes years of intensive exposure to develop a good micro-filaria-producer. Our men have not had that exposure.

3. In most of our men, the worms will probably all die before the man is desensitized; if some should survive, they will not be in large numbers, nor have sufficient life expectancy to produce secondary micro-filaria-carriers.

4. Transmission in the homeland will be inefficient also because of the average citizen's aversion to mosquitoes, and the extreme paucity of the reservoirs of micro-filariae.

5. Filaricidal drugs are known. They can be used if needed, to control transmission. At present, they are still in the experimental stage, and they appear to aggravate the symptoms of *mumu.*



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1. *Mumu* first appeared in the literature in an article by Prof. Buxton, entomologist of Samoa. In 1920 he described the phase of filariasis which we are now seeing. The word has become standard usage among all the troops who have been stationed in the Samoan area since Pearl Harbor It is used freely by Dickson et al[3] and Michael[3].
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3. Michael, Paul: Filariasis among Navy and Marine Personnel, U. S. Nav. Med. Bulletin 42:1059 (May) 1944.
4. Modified from Buxton, quoted by Dickson et al[3].
5. Personal communication, Lt. Comdr. W. P. Robert (MC), USNR.
6. Personal communication, Dr. Eric Fennel, Honolulu.

# Congenital Anomalies of the Coronary Arteries

LIEUTENANT COLONEL H. JULIAN FRACHTMAN, M.C., A.U.S.

As one would anticipate, congenital anomalies of the coronary circulation may take any conceivable form, varying from minor deviations to major defects. Similarly, the clinical manifestations of such anomalies may range from no symptoms at all, to marked coronary insufficiency incompatible with life. Abnormalities of this type are not of great importance to the clinical cardiologist; but to the pathologist, the pediatrician and the research cardiologist, they are worthy of note; and to the medical profession as a whole, because of the great importance of coronary disease, they should be of interest.

## EMBRYOLOGY

As the fetus develops its own circulatory system, there is in the beginning a single-chambered heart and a common efferent blood vessel, through which blood moves away from the heart. Later, endothelial buds branch off this common blood vessel to form the anlagen of the coronary arteries. A septum forms which divides the primitive single artery into the aorta and the pulmonary artery. Anomalous coronary arteries may result either from displacement of the endothelial buds or from displacement of the septum. Thus it is theoretically possible for both the coronary arteries to arise from the aorta, for both to arise from the pulmonary artery, or for the left or the right to arise from the pulmonary artery while the other takes its origin in the aorta.

## ANATOMY

The *left* coronary artery arises in the left posterior aortic sinus and after about 1 cm. divides into the *anterior descending branch* and the *left circumflex branch*. The anterior descending branch courses downward in the anterior longitudinal sulcus, overlying the interventricular septum, to the apex, giving off small branches to the adjacent portions of the left and right ventricles. The left circumflex artery runs in the coronary sulcus to the left, around the left ventricle to the posterior wall of the ventricle, usually terminating before reaching the posterior longitudinal sulcus. It thus nourishes the left atrium, the anterior and lateral portions of the left ventricle and a part of the posterior wall of the left ventricle.

The *right* coronary artery arises in the anterior aortic sinus and extends to the right around the right ventricle in the coronary sulcus. It gives off the *marginal branch* which travels along the right margin of the heart at the junction of the anterior and posterior surfaces of the right ventricle, and the *posterior descending branch* which follows the course of the posterior longitudinal sulcus toward the apex, where it may anastomose with terminal branches of the left anterior descending artery. The right coronary artery generally extends for a short distance after the posterior descending branch is given off to supply the right atrium and a portion of the left ventricle adjoining the posterior longitudinal sulcus. Thus, the right coronary artery nourishes the anterior, lateral and posterior walls of the right ventricle, the right atrium and a portion of the posterior area of the left ventricle.

## ANOMALIES OF THE RIGHT CORONARY ARTERY

The right coronary artery is *poorly developed* in eleven per cent of cases[1]. An instance of this was recently seen by the author in an individual who had died of an acute occlusion of the left anterior descending coronary. In spite of the fact that the right coronary was unusually small and poorly developed, there was no evidence of degeneration or fibrosis in the area normally supplied by it, indicating that the left coronary had taken over this function without difficulty by means of small branches. However, Whiting[2] reports the case of a 14-year-old girl who had frequently recurrent attacks of typical angina pectoris and who died following an emergency appendectomy. Post-mortem findings included a defective cusp of the mitral valve and a rudimentary right coronary artery. The heart weighed 425 grams, with a markedly thickened left ventricular wall (19 mm. in thickness) and a thin, atrophic right ventricular wall (3 mm. in thickness).

The right coronary artery may be entirely *absent*[3]. Krumbhaar and Ehrich[4] report the case of a 44-year-old woman who died from a pulmonary embolism. At autopsy, the right coronary artery was found to be completely absent and the single left coronary artery was found to supply the area of the heart normally supplied by the right. This was considered to be an incidental finding in view of the fact that there had been no evidence, during life, of cardiac disease. These investigators refer to 4 additional cases reported by other authors in which the right coronary was completely absent. The cases were: (a) a 37-year-old man who died as a result of subacute bacterial endocarditis, (b) a 33-year-old man in whom a cerebro-vascular accident was the cause of death, (c) a 39-year-old woman who died of pneumonia and (d) a 63-year-old man in whom carcinoma of the stomach caused death. In each instance the left coronary supplied the necessary branches to the usual distribution of the right coronary. Such an arrangement appears to be not incompatible with life.

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The right coronary artery may arise from the *pulmonary artery*. Soloff[5] in his survey of this condition notes two such cases. In each case, the individual was an adult who had had no previous cardiac symptomatology and who died of unrelated disease, one patient being 30 years of age and the other 63 years of age. At post-mortem examination, no significant pathological changes were found in the myocardium.

Abbott[6], in her study of congenital heart disease, mentions 10 cases in which the right coronary artery was found to arise from the pulmonary artery. No definite conclusions were set forth in this survey.

### ANOMALIES OF THE LEFT CORONARY ARTERY

Several instances of this congenital defect are reported in the literature. All possible variations in clinical manifestations occur, ranging from the complete absence of symptoms with incidental finding of the defect at autopsy to instances of extreme coronary insufficiency incompatible with more than two or three days of life.

Krumbhaar and Ehrich[4] cite an instance in which the left coronary artery was completely *absent*. The patient was a 35-year-old woman who died of carcinoma of the uterus with wide-spread metastases. She had had no cardiac manifestations prior to death. Examination of the heart at autopsy revealed the absence of the left coronary artery. The heart was otherwise normal, and weighed 285 grams. The area of the heart normally supplied by the left was supplied by branches of the right coronary artery. In their discussion, the authors refer to 5 similar cases reported by other sources. In each case the cause of death was an unrelated disease. Four of the 5 individuals were adults from 35 to 65 years of age and the fifth case was that of a child 3 years old. In all of these cases the absence of the left coronary was compensated for by the provision of branches by the normal right coronary artery to that part of the myocardium usually supplied by the left.

Origin of the left coronary artery from the *pulmonary artery* is reported by several authors.

Ruddock and Stehly[7] report the case of a 30-year-old man who died suddenly while doing hard work. He had previously been in good health without any cardiac symptoms. At autopsy, it was found that the left coronary artery arose from the posterior wall of the pulmonary artery. The distribution of the anomalous vessel was essentially normal. However, there was considerable communication between it and the right coronary artery, which arose in the normal position. The heart weighed 310 grams. The right and left ventricular walls were within normal limits for thickness.

Abbott[8] notes the case of a 64-year-old woman who died as the result of trauma. She had had no significant cardiac symptoms prior to death. On post-mortem examination the left coronary artery was found to arise from the pulmonary artery. The right coronary arose normally and followed the usual course, but was somewhat dilated. There was considerable anastomosis between the terminal branches of the right and left arteries. There were no other significant findings in the heart.

Chown and Schwalm[9] describe the case of an infant 5 months old. This child apparently had no symptoms which could be interpreted as angina pectoris but did reveal evidence of chronic disease in that there was a persistent pallor and inability to gain weight. Following broncho-pneumonia, the child expired. At autopsy, the right coronary was found to arise in the usual position, while the left arose from the posterior pulmonary sinus. The heart weighed 70 grams, though the normal weight at this age is about 30 grams. The coronary arteries followed a normal distribution. The wall of the left ventricle was from 6 to 8 mm. in thickness while that of the right was 3 to 5 mm. thick. Microscopic study revealed appreciable myocardial and endocardial degeneration. Myocardial failure was considered to be cause of death.

Soloff[5] presents the case of an infant 4½ months old who had seizures that were interpreted as being indicative of coronary insufficiency. At each feeding the child would apparently express severe pain by doubling up, drawing his feet up toward his chest, holding himself tense and motionless and becoming cyanotic. After a few moments he would vomit food, become very pale and perspire profusely. A few days before death it was noted that he became increasingly short of breath. Post-mortem findings included a markedly enlarged heart, 11 cm. in its transverse diameter and weighing 120 grams; the normal weight at this age is about 30 grams. The left coronary artery arose from the pulmonary artery while the right arose in its usual site. The distribution of these vessels was normal. The right ventricle showed no abnormalities while the left revealed an aneurysm of its lower half with very thin musculature.

Another instance of this defect was described in detail by Sanes and Kenney[10]. This was in a three-month-old female infant. Cough, dyspnea and emesis were outstanding symptoms. Peripheral edema and clubbing of the fingers were not observed. Cyanosis appeared only terminally. Physical examination revealed retraction of the intercostal spaces on inspiration, bulging of the left side of the chest, marked enlargement of the heart, poor heart tones, increased aortic second sound, and pulmonary atelectasis. At autopsy, the left coronary artery was found to arise from the pulmonary artery, while the right arose normally. The left ventricle was hypertrophied and dilated. There was a partial apical aneurysm present. Considerable myocardial degeneration, necrosis and fibrosis of the left ventricle were noted.

These authors (Sanes and Kenney) also reviewed cases reported separately by Abriskoff, Heitzmann,

Kiyokawa, Corrington and Krumbhaar, and Scholte. The patients were respectively 4, 3½, 4, 10 and 2½ months old, and in each, the left coronary artery arose from the pulmonary artery. The clinical and pathological findings in each case were similar to those in the case just described.

Another instance of this anomaly is recorded by Bland, White and Garland[11]. A few others have been reported by German investigators. In this group of cases, the ages of the patients varied from two days to fifty-three years at the time of death.

### MISCELLANEOUS ANOMALIES

Origin of both coronary arteries from the pulmonary artery is extremely rare. This defect is not compatible with life. Grayzel and Tennant[12] describe such a defect in an infant who died at the age of ten hours. Ruddock and Stehly[7] refer to a case reported by Limbourg of an infant that died at the age of ten days. In each of these, both coronary arteries were found to arise from the pulmonary artery.

Variations in the distribution of coronary arteries that arise in the usual site occur very commonly. However, the normal range for such anatomical variations is wide and they appear not to disturb the physiology of the heart. Anastomosis between terminal branches of the right and left coronary arteries, and the distribution of many small unnamed branches, compensate well for any such departures from the classical anatomy.

One or more accessory coronary arteries may be present, but this is of no practical importance.

Anomalies of the venous circulation of the heart, and the persistence of the embryonic sinusoids, are of no significance.

There are no reported cases of the absence of both coronary arteries.

### DISCUSSION

Probably the outstanding lesson to be emphasized from the study of these anomalies is the fact that adequate arterial anastomosis is the basis for proper nutrition of the heart and consequent normal function. Hyrtl's postulate that one artery should supply the whole heart appears to be borne out in many of these cases. The absence of clinical manifestations in several instances of anomalous cardiac blood supply can best be explained on the basis of direct arterial or arteriolar connections between the normal and abnormal vessels, or in the case of an absent vessel, by the presence of branches from the remaining artery supplying the area of the heart normally supplied by the artery that is missing. Acceptance of this premise aids in understanding the variation in mortality in acute coronary occlusion in hearts which have the usual anatomy of the coronary arteries. The individual who lives through such an episode has developed a new blood supply to the area of infarction while the individual who dies has failed to meet this demand.

In the same manner, the development of compensatory blood supply in the heart in which the coronary circulation is congenitally abnormal permits the heart to function in a normal manner. The cases cited, of adults who have lived for many years without clinical manifestations of such defects, substantiate this thesis. On the other hand, cases of a grossly similar type have demonstrated incompatibility with life for more than a few days or a few months. In these instances, compensatory anastomoses have failed to develop. The extent of the myocardium supplied by the anomalous artery and the degree of exertion by the infant may possibly determine the average length of life.

The influence exerted by the amount of pressure and degree of oxygenation in the coronary blood flow should be considered. Deficiency of oxygen in this circuit appears to be a most important factor. Low oxygen tension leads to accumulation of lactic acid and other irritant metabolites. This in turn brings about the pathological changes of degeneration, necrosis and fibrosis. These findings are similar to those found in the hearts of adults in which there has been gradual coronary occlusion.

But in the final analysis, anastomosis would seem to be the key to the problem, for it alone can explain why some individuals with the same anomalous origin of a coronary artery live for many years without cardiac symptomatology while others die after only a few months of life. If there is adequate anastomosis, the problems of oxygenation and pressure are readily solved. This, of course, would not be true in those extremely rare cases in which both coronary arteries arose from the pulmonary artery.

As far as ante-mortem diagnosis is concerned, this can be done in some instances, but, in general, it is the pathologist who will find these defects.

### SUMMARY AND CONCLUSIONS

1. Congenital anomalies of the coronary circulation occur very infrequently.

2. Instances are reported of the absence of either of the coronary arteries, or of the origin of the left or the right or of both coronary arteries from the pulmonary artery.

3. The clinical manifestations in these various defects range from no apparent symptoms or signs to incompatibility with life.

4. Except for those defects which are obviously incompatible with life, such as the origin of both coronary arteries from the pulmonary artery, no definite correlation can be made between the gross anatomical defect and the absence, presence or degree of signs and symptoms of coronary insufficiency.

5. In those cases in which clinical evidence of the defect is lacking, it would appear that compensatory circulation has developed. When such compensation has not occurred, coronary insufficiency is manifest.

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# Gastrojejunal Ulcer as Related to Gastro-Enterostomy

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Over a period of twenty-five years, during which time I have been actively interested in the surgical treatment of lesions of the stomach, there has been a radical change in the atttitude regarding the value of gastro-enterostomy.

As early as 1916, Bland Sutton of England began calling attention to the fact that gastro-enterostomy in the treatment of nonobstructing duodenal ulcers gave unsatisfactory results, and advocated pylorectomy with removal of the ulcer. The German surgeons, particularly von Haberer, wrote voluminously on this subject and advised even more radical surgery, namely, removing as much as one-half of the stomach.

During the early 1920's, in America, gastro-enterostomy with or without modification was enjoying great popularity and was almost universally regarded as the operation of choice in the treatment of benign lesions of the stomach and duodenum. Such was the case as noted in various clinics on my trips to the mainland in the early '20's with one exception. At Mt. Sinai Hospital in New York on the service of Drs. Lewisohn and Berg, radical removal of the stomach was being done for both benign gastric and duodenal ulcers. Well do I remember Dr. Lewisohn presenting a paper before the New York Academy of Medicine in which he stated that they had found an incidence of 34 per cent of gastrojejunal ulcers following gastro-enterostomy, 18 per cent proven by re-operation, this being the basis for their discarding conservative operations for radical resection. The discussions that followed this presentation were heated and without exception discredited Dr. Lewisohn's observations.

Fig. 1. X-ray of stomach after gastro-enterostomy and previous to resection.

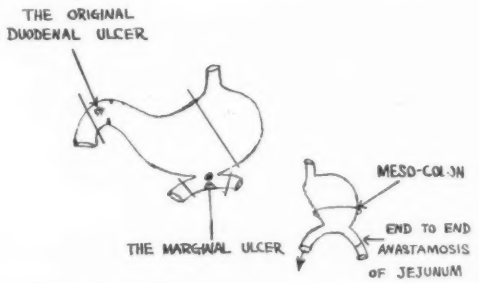


Fig. 2. Technique of dealing with marginal ulcer. Radical resection of stomach with limbs of gastro-enterostomy, end-to-end anastomosis of jejunum, anastomosis of jejunum and remaining stomach, Polya-Hofmeister method.

It has been interesting to me to follow the trend of events as related to this subject since that time. Dr. Lahey, an original believer in conservative surgery for such lesions, pointed out that it was hard to convince the medical profession that it was necessary to sacrifice a large amount of apparently normal stomach to cure a lesion of the duodenum frequently no larger than a few millimeters in diameter. With few exceptions, in America as elsewhere, it is now the belief of those with the greatest experience that the greatest success in dealing with gastroduodenal ulcers follows radical resection of the stomach. No one claims that gastrojejunal ulcer never follows radical resection, for it occasionally does. It is maintained, however, that gastric acidity is reduced by resection to the point where this likelihood is small.

With present methods of pre- and postoperative care; with improved types of anesthesia; with adequate amounts of blood at our disposal in the prevention of shock, gastric resection can be done with comparative safety. Considering the complications that are likely to follow, and operative mortality, radical resection is the operation of choice, in preference to conservative operations, in the majority of cases.

Gastro-enterostomy in the treatment of duodenal ulcer may be the operation of election if there is marked pyloric obstruction, if the individual is beyond middle life and the gastric acidity is relatively

low, and particularly if, added to this, the individual is a poor operative risk.

The subject of gastric resection for duodenal ulcer has recently been well presented by Dr. Lewisohn in the April issue of *Surgery, Gynecology and Obstetrics* for the current year.

Early in my experience with the surgical treatment of duodenal ulcer, I became convinced that radical resection was the operation of choice, and I have not, without special indications, done conservative operations for a number of years.

Fig. 3. Specimen removed at operation—stomach and first part of duodenum with attached limbs of jejunum leading to gastro-enterostomy.

However, in my more immature years, to be exact in 1920, just twenty-five years ago, I did do a gastro-enterostomy on an individual, a Filipino man, age 20, for duodenal ulcer. Subsequent developments have been interesting and informative. For a year or two he was relieved of his symptoms, but on the whole he continued to have epigastric distress. In September of 1944 he was admitted to The Queen's Hospital because of a rather severe gastric hemorrhage. Subsequent roentgenograms and gastroscopic examinations confirmed our impression that the patient had developed a gastrojejunal ulcer and this was verified at operation. Uneventful recovery followed radical gastric resection.

Gastrojejunal ulcer is a serious complication. It is distressing to the patient; it seldom responds to medical measures; it is frequently associated with serious hemorrhages; and it may perforate into the abdomen or into the transverse colon, forming a gastrojejunocolic fistula. The most characteristic clinical finding is that whereas the duodenal ulcer produced epigastric pain and distress, the discomfort is now to the left and above the umbilicus at the site of the gastrojejunal stoma, and in this area there is usually marked tenderness. Visualizing the ulcer crater may or may not be possible with x-ray studies, and the same may be the case with the gastroscope. Both methods are usually diagnostic, however.

I have had occasion to operate upon 13 such complications, all following gastro-enterostomy for duodenal ulcer. One had perforated into the colon and another necessitated resection of part of the wall of the colon in its correction. The onset of a colic fistula introduces a number of complicating features that are fraught with grave danger to the patient, and the correction of this condition is a major surgical event. When the colon is not involved, radical resection of the stomach and ulcer is carried out as shown in the accompanying diagram.

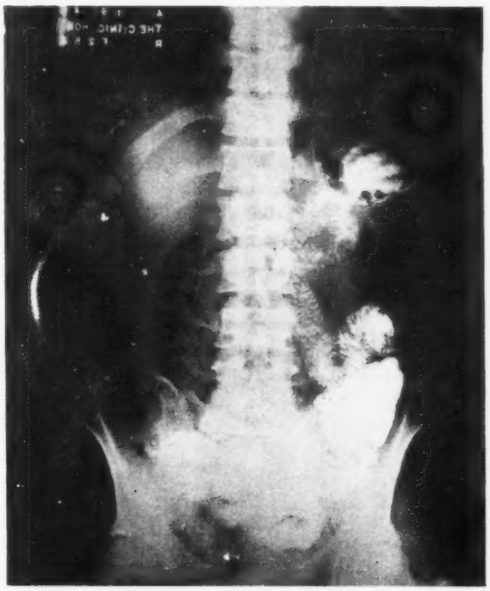

Fig. 4. X-ray to show small amount of stomach remaining.

## SUMMARY

1. Radical resection of the stomach, when surgical intervention is indicated in duodenal or gastric ulcer, is the operation of choice.

2. Gastro-enterostomy in benign ulcer may occasionally be indicated in selected cases.

3. Gastrojejunal ulcer not infrequently follows gastro-enterostomy for duodenal ulcer but may not develop for as long as twenty-five years following the primary operation.

# Bronchiectasis

COLONEL FORRESTER RAINE, M.C., A.U.S.

Bronchiectasis is hardly a disease entity, because it arises as a result of, or is a complication of, so many different diseases. Actually, bronchiectasis means "dilation of part or virtually all of the bronchial tree." With this enlargement is associated some change in the mucous membrane, frequently ulceration; some change in the bronchial wall, usually loss of elastic fibers; and very commonly there is complete absence of a part of the bronchial wall with the bronchiectatic space extending into the surrounding lung tissue.

Bronchiectasis may be divided into congenital and acquired types. Either of these types may take forms of bronchial dilation which we describe as saccular, cylindrical, fusiform, or varicose.

## CONGENITAL BRONCHIECTASIS

Bronchiectasis has been called a children's disease, and in many respects this is true, for it tends to be present or start early in life, although it may not be recognized until adult life. The congenital form of bronchiectasis is the result of developmental anomalies; it may involve only a portion of the bronchial tree, which gives rise to the telangiectatic form, or it may involve virtually the entire bronchial tree, the universal type. Congenital cystic disease has been classed by some as a form of bronchiectasis, but this concept is open to some question.

Symptoms of pulmonary or bronchial involvement may be absent in the newborn in spite of rather extensive bronchiectatic lesions. Their presence becomes apparent only after an upper respiratory infection when extension involves cavities or one of the independent cysts ruptures producing empyema or tension pneumothorax.

## ACQUIRED BRONCHIECTASIS: CAUSES

Several causes of bronchiectasis are known, but there are many patients the etiology of whose disease is obscure. Causative agents may be grouped as follows:

### *Intrabronchial*

Foreign bodies and bronchial tumors both benign and malignant are common causes. The mechanism of the development of bronchiectasis from partial obstruction of the bronchial lumen is rather easy to understand. Infection of the bronchial wall occurs as a result of the obstruction. Cough, with increased intrabronchial pressure on the wall weakened by infection, results in dilatation of the bronchi. It is known that this process may occur rather rapidly, far advanced bronchiectasis having been found distal to a foreign body in the bronchus as early as three months. Besides the bronchiectasis which develops, there may be, of course, associated lung abscess.

### *Bronchial*

Changes in the bronchial wall causing bronchiectasis are not well understood and to a certain extent these factors are still in the hypothetical stage. Presumably metaplasia of the mucosa can cause partial obstruction; certainly ulceration of the mucosa with resultant contracture by scar can cause bronchial stenosis and bronchiectasis. There is possibly some relationship between hypersecretion of the bronchial mucosa and bronchiectasis, but known cases of severe bronchitis have existed for years without the development of any bronchiectasis. This is even true of putrid bronchitis, which at times produces large amounts of foul-smelling pus similar to that found in bronchiectasis.

### *Peribronchial*

Enlarged lymph nodes, tumors or aneurysms may compress the bronchial wall, producing a partial obstruction with resultant bronchiectasis.

### *Pulmonary*

Under this heading may be grouped several types of factors, some of which are known to produce bronchiectasis, while others are presumed to. It is thought that a goodly number of cases of bronchiectasis which are discovered without known specific cause, have been the result of pneumonia or influenza, particularly the pneumonia associated with measles or whooping cough. Certainly a large number of patients, both in childhood and adult life, are found with a far-advanced bronchiectasis for which no specific mechanical factor can be found. A history of pneumonia in childhood, of a severe whooping cough, or of protracted illness with measles, can frequently be elicited, and it is presumed that bronchiectasis has developed as a result of these diseases. Lung abscess, tuberculosis, pulmonary fibrosis, and emphysema frequently have bronchiectatic lesions associated with them. Bronchiectasis here develops as a result of pressure, erosion, or ulceration of the bronchial wall with resultant obstruction and bronchial dilatation. Unresolved pneumonia was for years thought to be a prime cause of bronchiectasis, but there is considerable question in my mind whether there is such an entity. It seems more likely that the

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initial lesion may have been pneumonitis with involvement of the bronchi, or wet atelectasis resulting in bronchiectasis or lung abscess, or both.

Until very recently it was felt that bronchial dilatation once established remained so. Very little reduction in the size of dilated bronchi has ever been observed under any type of treatment. Blades has described some rather marked bronchial dilatations following atypical pneumonia which have apparently receded completely in the course of two to three months. It is interesting to note that bronchoscopic observation revealed a very different picture from that of a real bronchiectasis. The mucous membrane was not friable and did not bleed easily as it does in the bronchus draining bronchiectatic cavities.

## Symptoms of Bronchiectasis

Bronchiectasis may be unilateral or bilateral, and commonly involves the lower lobes. It becomes apparent clinically most frequently between the ages of 10 and 40. The primary symptoms are cough, expectoration of foul-smelling sputum, hemoptysis, and fever. Cough may be severe and almost uncontrollable. In some cases, on the other hand, cough is relatively slight. Expectoration is almost always present, but there are some types of dry bronchiectasis wherein expectoration may be extremely scanty. The sputum is usually abundant and foul-smelling. It may vary in amount from 30 to 1500 cc. in twenty-four hours. Hemoptysis is a very common symptom of bronchiectasis, occurring more frequently than in tuberculosis. Bleeding may be slight, continuing in small amounts for weeks. It may be massive, and many patients have died from hemoptysis. Fever is an inconsistent symptom but almost all cases will have recurring bouts of fever as the result of the bronchial wall infection extending into the surrounding pulmonary tissue. A history of repeated attacks of pneumonia should make one suspicious of bronchiectasis as the underlying lesion.

## Diagnosis

Bronchiectasis can be diagnosed from the history and symptoms alone, but the diagnosis should include the type of bronchial enlargement and its precise location. This can be done only by the combined use of bronchoscopic investigation and lipiodol instillation. Furthermore, single anterior-posterior x-ray films of lipiodol instillations will not give the whole story. When only one side is filled a lateral plate will suffice, but when both sides of the bronchial tree are filled a right and left oblique view must be taken. Differentiation can be made as to the involvement of the middle or lower lobes on the right, and between the lower lobe, and the lingual portion of the upper lobe, on the left.

Physical findings vary tremendously, depending upon the amount of secretion in the bronchi and whether at the time of examination there is associated pulmonary involvement. Coarse rales may be heard; there may be evidence of areas of atelectasis or even suggestions of cavities. Clubbing of the fingers may commonly be found. The mechanism of the production of clubbed fingers is still not entirely understood. We know that it can occur very rapidly, for I have seen it appear in the amputation stump of a finger in the course of three months. Furthermore, it is to some extent reversible, in that correction of the underlying lung pathology will cause recession of the clubbing if it has not existed too long. Bronchoscopic inspection of the bronchial tree is important in determining the origin of the expectoration, or the presence or absence of a foreign body or of obstruction to the involved lobe or lobes.

## Accessory Nasal Sinus Disease

The relationship of chronic infection of the nasal sinus to bronchiectasis is not entirely clear. Certainly associated nasal sinus disease aggravates the bronchial infection, for many patients will have marked diminution of their symptoms after eradication of the sinus infection.

## Treatment of Bronchiectasis

As in other diseases, prevention should be of major concern. The following factors which are known to produce bronchiectasis should be eradicated or corrected as soon as possible. Foreign bodies in the bronchial tree should be removed immediately. Atelectasis should not be allowed to persist, and if the simpler methods of overcoming it by postural drainage, by encouraging cough, or by forced breathing of carbon dioxide are not sufficient to clear the bronchial tree and permit complete aeration of the involved lobe or lobes, then the secretion should be aspirated bronchoscopically. Bronchial stenosis, when amenable to treatment, should be cleared as soon as discovered. A benign bronchial tumor causing obstruction should be removed bronchoscopically. Lung abscesses should be treated adequately as soon as possible, since persistent lung abscess is known to produce bronchiectasis.

Evolution in treatment has been rapid, and a great many of the changes have occurred within the last twenty years. It will be worthwhile, I think, to review this evolution so that we may have a better understanding of treatment as it stands today.

### *Rest*

Rest in bed, although indicated during remissions of the disease, does not cure bronchiectasis. Symptoms will be relieved and temporary improvement will occur, but no lasting improvement results. Many doctors have felt that changes in climate are beneficial, and it does seem that some patients have much less expectoration and are more comfortable in a

high, dry climate; but climate per se does not cure the disease.

### *Postural Drainage*

Postural drainage was one of the early forms of treatment. Time has established its position as of prime importance in the treatment of the disease. Postural drainage should be tried in every case before other forms of more radical treatment are considered. Drainage may be carried on continuously or at intermittent periods. Many patients, if drained adequately twice a day, can be kept free from appreciable cough at other periods. The fetor of the sputum diminishes, sometimes markedly; recurring bouts of infection are less frequent and less severe; and hemoptysis may cease.

### *Medication*

Drugs of all kinds and descriptions have been tried, but none of them is of any lasting benefit. Arsenicals have been used, and many believed for a time that the fetor was diminished by them, but no known cures have occurred.

Bronchial sprays of all types of drugs have been used as air saturation in a closed room or by direct bronchoscopic application, but none of them have been appreciably beneficial. The instillation of lipiodol was in vogue for a considerable time, and it was felt by many that lasting benefit resulted, but this is open to grave question.

It has been difficult to understand the apparent improvement in some patients following lipiodol instillations. There is considerable absorption of iodine, but iodine therapy, per se, does not produce appreciable benefit. The soothing effect of the oil on inflamed mucous membranes may account for some of the temporary improvement noted. At the present time it does not appear that this treatment is beneficial.

### *Bronchoscopic Aspiration*

Bronchial aspiration, without question, accomplishes relatively complete evacuation of the secretions, and by shrinking the hypertrophied mucous membrane of the involved bronchus permits optimal evacuation of secretion by postural drainage. This improvement in drainage may last for a considerable time before repetition of the shrinkage with aspiration is necessary. Bronchoscopists tend to be enthusiastic about the continued and permanent results of bronchoscopic aspiration, but it is felt that although it is a distinct aid in improving the results of postural drainage and that it is essential to proper diagnosis, it is not a cure for the condition.

### *Collapse Therapy*

Three primary types of collapse therapy have been used. It was thought early in the evolution of treatment that pneumothorax would afford collapse of the diseased bronchi, permit them to heal, and thus cure the disease. Unfortunately, one can secure a beautiful collapse of the entire lung and still have bronchial dilatations the same size as they were before. Paralysis of the diaphragm was thought for a time to be very beneficial. Symptoms were commonly relieved and it seemed that hemoptysis was reduced in amount and frequency. Continued observation, however, has proven that paralysis of the diaphragm, per se, is not of benefit in bronchiectasis. Because of apparent benefit from both pneumothorax and paralysis of the diaphragm, thoracoplasty for permanent collapse of the involved lobes was tried. Here again, continued observation showed that the bronchi did not change. They remained just as large and had just as much ulceration as before the operation. Collapse therapy, therefore, has been abandoned. A few other types of operative treatment such as ligation of the pulmonary artery to the involved lobe, incision and drainage of lung and bronchi, compression by tamponade, all have been tried. None of them worked.

### *Lobectomy*

Since it was impossible, apparently, to collapse the diseased bronchi, or cure them by intravenous therapy or direct application of chemicals, removal of the involved lung became the goal of treatment. Occasionally, through the first twenty years of this century, lobectomy had been performed for the cure of bronchiectasis; but the mortality rate was so appalling that the operation was abandoned. Beginning immediately after the last war, new attempts were made, and many changes in technique were devised in the endeavor to remove all the diseased lung without a prohibitive mortality. During this period Graham developed his cautery pneumonectomy method and succeeded in relieving many of these patients of the bulk of their diseased lung and bronchi without a prohibitive mortality. The operation, however, was time-consuming, tended to be somewhat deforming, and resulted in many persistent bronchial fistulas which, although a great improvement over their preoperative condition, was not an ideal solution. Alexander developed his two-stage lobectomy with mass ligation of the lobe causing slough of the lobe with, of course, local empyema and bronchial fistula. Most of these fistulae healed and his results were rather good. Whittemore developed an exteriorization operation with combined thoracoplasty which was effective in removing the greater part of the diseased tissue without a prohibitive mortality. Shenstone, Archibald, Brunn, Coryllos, Lilienthal, and many others contributed variations in technique which gradually reduced the operative mortality and resulted in a high percentage of cures.

During this period of development, pneumonectomy for carcinoma rapidly came to the fore and the technique improved by leaps and bounds. It soon

became apparent that individual ligation of the vessels with meticulous closure of the main bronchial stem resulted in primary healing in a large percentage of cases. Furthermore, thoracoplasty was not necessary even in most instances of total pneumonectomy. Empyema did not necessarily develop and bronchial fistulas became only an occasional complication. This success of individual ligation and meticulous closure of the bronchus led several surgeons to try the same sort of technique for lobectomy. Rienhoff and Blades were among the early ones to work out the anatomical details and operative technique. Under this technique, lobectomy in a single stage operation with primary healing has become the rule rather than the exception. Thoracoplasty is not necessary: the remaining lobe or lobes expand and fill the chest cavity. Mortality rates have diminished so that now it is a relatively safe operation.

I shall not discuss the technique of the operation. You are interested, however, in determining which patients should be referred to a thoracic surgeon for treatment. Certainly, not all patients with bronchiectasis should be operated upon. If a patient by practicing postural drainage can remain comfortable and free of fetor, recurrent hemoptysis and fever, operation would not seem indicated. Their bronchiectasis will never be cured but at the same time it may never progress sufficiently to warrant lobectomy.

It is impossible to predict in many patients what the course of their disease will be. We know that some will continue, without appreciable change for the worse, over a period of several years. Others will tend to go downhill rather rapidly. Children with associated pneumonitis and the development of fibrosis do badly. The obnoxious fetor associated with bronchiectasis will at times almost disappear under postural drainage, while in other patients there will be so little improvement that it is negligible. When continued infection in bronchial wall persists in spite of attempts at drainage, hemoptysis is likely to become a serious symptom.

The complications of bronchiectasis occur fairly frequently. Suppurative pleurisy is not infrequent. It is quite likely to be an encysted empyema. Amyloid disease occurs after long continued bronchiectasis. It is not different from that which follows other types of chronic infection. Moderate amyloid disease is not necessarily the contra-indication to surgery many have believed, for I have seen moderately severe cases improve markedly after the infection is eradicated.

Brain abscesses are prone to follow bronchiectasis just as they do any suppurative disease of the lung.

### *Indications for Lobectomy*

Patients who cannot be kept reasonably free of symptoms by occasional bronchoscopic aspiration and postural drainage should be operated upon. These persistent symptoms which are indications for operation are as follows:

a. Those whose fetor and foul-smelling sputum does not diminish to a point where they are not obnoxious to themselves and others.

b. Those who continue to have hemoptysis, either persistent small hemorrhages or occasional massive hemorrhages.

c. Those who have recurring attacks of fever which indicates extension through bronchial wall into surrounding lung. Many of these patients have associated small-lung abscesses which never drain adequately and therefore never heal completely.

The presence of tuberculosis at one time was considered an absolute contra-indication to lobectomy, but it is felt now that bronchiectasis in the tuberculous, resulting from bronchial stenosis, can be benefited very materially by lobectomy, although their chances for getting primary healing of their bronchi are not quite as good as those of the non-tuberculous. Primary healing is being achieved with increasing frequency, however.

The treatment of bronchiectasis is not yet ideal, but great strides have been made in the last 20 years. Many patients are today entirely symptom free who were, prior to operation, practically outcasts from society because of their bronchiectasis.

# Diagnosis and Treatment of Lung Abscess and Acute Empyema

COLONEL GEORGE FINNEY, M.C., A.U.S.

Discussion of the two conditions of lung abscess and acute empyema is rather hackneyed to many. However, the continued high mortality rate of the former, and the fact that chronic empyema still all too often follows an acute empyema, justifies a review of their diagnosis and treatment.

## LUNG ABSCESS

In the discussion of lung abscess, no separation will be made between cases of pulmonary gangrene and abscess, because in the majority of cases, there is no clear-cut dividing line. There is a certain amount of necrosis or gangrene associated with almost all lung abscesses sooner or later. No consideration will be given to abscesses complicating either neoplasms or bronchiectasis.

### *Incidence*

Lung abscess may occur at any time of life, but it is much more apt to occur in males; in some reported series the ratio of male to female cases has been as high as 2:1.

The duration of the abscess is of great importance. In analyzing a number of reported series of cases, it can be seen that many patients recover spontaneously, but the percentage of unsuccessful results increases rapidly with the increase in elapsed time from the onset of symptoms until proper treatment is instituted. There seems to be a general belief that most lung abscesses occur in the lower lobe. This, however, is not borne out by reports of numerous observers.

### *Etiology*

Time will not permit a full discussion of the etiology of lung abscess. However, the two main schools of thought have been based on aspiration and embolism, respectively, as initiating the first step in the formation of an abscess. There are considerable data to support evidence that each does play some part. However, the most important single step in the pathogenesis of the majority of lung abscesses is atelectasis. This may occur in the course of pneumonia if bronchi are plugged with thick exudate or post-operatively if a bronchus is plugged with blood and mucus or foreign material such as vomitus. Under these circumstances, if there are virulent bacteria blocked in the atelectatic lung, abscess is likely to result.

The bacteriology of lung abscess is still not clearly understood. The only definite thing that available evidence indicates is that there is no one organism which is primarily responsible. Pyogenic organisms of the aerobic and anaerobic type, as well as spirochetes and fusiform bacilli, can all be grown from the sputum and pus. It seems quite likely that this symbiosis is important, because in order to produce chronic lung abscess in experimental animals, it has been necessary to use several different types of bacteria, or bacteria and spirochetes together, in the inoculum.

### *Diagnosis*

The outstanding clinical signs and symptoms of lung abscess are:

1. Cough is present in practically all cases.
2. Pain is present in at least 75 per cent of cases, often early.
3. Fever occurs in more than 70 per cent of cases.
4. Foul sputum is present in nearly 80 per cent and in half of these it may be bloody at times.
5. Physical signs may be characteristic of cavity but in a large number of cases there may be *no* pulmonary signs whatever.
6. X-ray usually reveals an area of increased density at first, then a sharper demarcation followed by a cavity with fluid level.

### *Prophylaxis*

Before we consider the treatment of lung abscess a word should be said about the importance of the preventive aspect of therapy. Because abscess develops in many individuals who have never had proper oral hygiene, it is imperative that proper care be taken to prevent aspiration of foreign material during any surgical procedure. This is particularly true of both surgical and dental operations about the mouth or throat. The simple expedient of raising the foot of the patient's bed and making sure that the bronchi are kept clear by frequent suction with a nasal catheter will prevent many potential lung abscesses. Hyperventilating each patient with carbon dioxide and oxygen at the end of every surgical operation will practically eliminate the possibility of atelectasis and so prevent the stagnation of mucus. A good anaesthetist who is alert to the dangers of lung abscess can thus institute the best prophylaxis. In those post-operative cases of massive collapse of the lung where signs indicate a mucus plug is present causing atelectasis, its immediate removal by bronchoscopy will not only afford necessary relief to the patient but also possibly prevent the formation of an abscess.

### *Treatment*

The treatment of lung abscess naturally falls into either conservative medical therapy, or surgical drainage if the former fails. Because of the fact that a

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reasonable number of acute abscesses will heal spontaneously if given a fair trial under an adequate conservative program, this should always be done. When the diagnosis has been established, a comprehensive medical regimen of bed rest and high caloric, high vitamin diet, together with steam inhalations containing creosote and benzoin, should be instituted. Ephedrine and other anti-spasmodics by mouth can be very helpful at times. Early postural drainage is of paramount necessity. Experimentation with different positions will soon show which is most effective in emptying the abscess. It is usually easiest for the patient to be over the side of the bed with his hands on a low chair, with body turned so that the affected side of the lung is highest. Vigorous coughing should be avoided as far as possible. The postural drainage should be carried out at regular intervals, starting with five minutes and gradually increasing the length of time to ten or fifteen minutes, depending on the patient's condition. Potassium iodide or ammonium chloride by mouth can often be used to advantage, but it has been pretty well shown that there is no drug that is in any way specific, because of the mixed bacterial flora. There are a few cases, where spirochetes are present in large quantities, that will react very favorably to the arsenicals if these are given early. Also, the use of the sulfonamides and penicillin in the early stage of pneumonitis will undoubtedly abort many potential abscesses. However, when a true abscess is present, no benefit can be expected from these drugs*. Whole blood transfusion, particularly in those cases where there is a secondary anemia, can be very valuable and should be used. Bronchoscopy is important for diagnosis and also for ruling out the presence of a neoplasm or foreign body, but its therapeutic value for drainage alone is questionable. There are cases when drainage will be improved by the removal of obstructing granulations and shrinking of the mucous membranes. However, if there is not definite benefit and the patient does not tolerate bronchoscopy well, it should not be persisted in.

In cases of lung abscess where surgical intervention becomes indicated, the procedure of choice in most surgeons' hands is external drainage. This is usually best carried out in two stages. Accurate localization of the abscess is of paramount importance. When this has been done, rib resection with exposure of a fairly wide area of pleura is accomplished and the wound packed open. The area of pleura exposed should be immediately overlying the abscess. The second stage operation is usually carried out after forty-eight hours to insure adhesion of the parietal to the visceral pleura. Then when the abscess cavity is entered, using the actual cautery, there will be no danger of contamination of the main pleural cavity and adequate external drainage can be established. The temptation to put a needle in the chest before the operation to help localize an abscess should be withstood, because of the great danger of spreading infection. There are a few cases where lobectomy for abscess is indicated and when this is carried out in competent hands the results are very satisfactory. However, such cases are in the minority. The use of pneumothorax or phrenicectomy is mentioned only to be deprecated.

There is still considerable controversy regarding the time when surgical drainage should be instituted. This requires the careful consideration and keen judgment of both the physician and the surgeon. In general, it is felt that if a proper medical regimen has been carefully carried out for a period of three weeks without showing a definite satisfactory response, surgical intervention is indicated. When we examine reports of the treatment of lung abscess from various clinics we find that the percentage of unsatisfactory results rises rapidly as the duration of treatment goes beyond six weeks. It should also be noted that peripherally located abscesses are less apt to clear up under medical management and are more amenable to operation. Surgery should therefore be resorted to earlier in the treatment of this type of abscess than in the centrally located ones, in which the medical regimen is more apt to prove effective and surgery more hazardous.

As stated earlier, results in the treatment of lung abscess as a whole are still not satisfactory. It should be noted that Neuhof has been an advocate of early surgical intervention and undoubtedly surgical intervention has been put off too long in many cases.

## ACUTE EMPYEMA

Since the advent of sulfonamides and penicillin the incidence of acute empyema has been cut down considerably. However, acute empyema still occurs often enough to concern both the physician and surgeon in its diagnosis and proper treatment, with a view to reducing the morbidity and the number of chronic empyemas that may develop.

### *Etiology*

There are, in general, two main types of acute empyema. There is the localized pocket or encapsulated type, usually caused by the pneumococcus, secondary to pneumonia. This pocket may be peripheral at some point against the parietal pleura or it may be interlobar or against the mediastinum. Then there is the massive type. This may be a diffuse pleuritis causing the formation of large quantities of fluid with very little fibrin, as is usually the case when the infecting organism is the streptococcus. Postpneumonic pneumococcal empyema may also be massive, with very thick pus containing large amounts of fibrin. An empyema resulting from a wound of the thorax will be as a rule of the massive type, particularly if a hemothorax becomes infected.

Acute empyema may occur at any time of life, and there is no appreciable difference in its incidence in the two sexes. The most common etiologic factor in

*Recent reports indicate favorable results from penicillin in aerosol form.—ED.

civilian practice is of course lobar pneumonia, while in war surgery secondary infection occuring in wounds of the chest may be (though it seldom is) responsible for the development of empyema.

The bacteriology of acute empyema is well understood and is of considerable importance. This is true both in its relationship to prognosis and also because the type of surgical treatment and the time it should be instituted depends to a great extent on the type of infecting organism. The most common organism found in cultures of empyemas is the pneumococcus. The streptococcus is present in pure culture in a certain number of cases. However, particularly in empyemas developing as the result of trauma, a mixed flora may be present and there may be both aerobic and anaerobic types. Further consideration will be given to the bacteriology when treatment is discussed.

### *Diagnosis*

The outstanding clinical signs and symptoms of acute empyema are:

1. Development of a septic type of fever.
2. Dull to flat percussion note with diminished or absent tactile fremitus.
3. Distant or absent breath sounds.
4. Limitation of respiratory excursion on the affected side together with displacement of the mediastinum toward the opposite side.
5. X-ray findings may be characteristic of fluid, but many times, in those cases where the clinical signs of fluid are equivocal, x-ray findings are likewise.
6. The aspirating needle is the best means of making a positive diagnosis; it should be used in every case where fluid in the chest is suspected.

### *Prophylaxis*

Just as the preventive aspect is so important in the consideration of lung abscess,. so also is this true in acute empyema. Adequate chemotherapy when instituted early in the pneumonias has without doubt cut down the incidence of acute empyema to a marked degree. Also its prophylactic use together with large doses of penicillin in various types of thoracic war wounds has prevented secondary infection to such an extent that very few hemothoraces develop into empyema. Also, large wounds of the thorax can be closed and treated conservatively with very little danger of secondary infection. By the combined use of the sulfonamides and penicillin many potential empyemas have been aborted.

### *Treatment*

Much has been written about the treatment of acute empyema in the past. For the most part the discussion revolved around the question as to whether some form of closed drainage of the empyema had a lower mortality rate than open drainage with rib resection. This question, like so many others of its type, has not been answered to the satisfaction or agreement of all surgeons. However, it has been demonstrated that mortality rates depend more on the virulence of the infecting organism and the presence of further complications rather than the type of surgical drainage employed, provided the drainage is adequate. There are certain principles that must be followed in order to obtain the maximum of results.

In the first place, the empyema that proves to be a surgical emergency is the exception. Aspiration of the cavity will not only serve to relieve respiratory embarrassment if that is present, but it will also allow culture of the fluid so that the bacteriology can be definitely determined. In a certain percentage of cases, particularly where the hemolytic streptococcus is the infecting organism, repeated aspirations together with proper chemotherapy will give excellent results. However, the chances of effecting a cure in pneumococcic empyemas by repeated aspirations, even though adequate chemotherapy is being carried out, are not good. Therefore, aspirations alone as surgical treatment should be repeated only until thick pus has developed provided the fluid did not clear up entirely and disappear. When frank pus is present adequate drainage should be established. As a result of using both closed drainage of some type in certain cases and open drainage with rib resection in others, the following principles of treatment have been established. In encapsulated empyemas, and particularly in those that are in an interlobar space, open drainage with rib resection is the treatment of choice. In massive empyemas, unless there are anaerobic organisms present, some form of adequate closed drainage is usually preferable. The word adequate is stressed because simply inserting a rubber tube in a cavity and expecting it to drain properly is not adequate. In those cases where there is an anaerobic infection, open drainage should be instituted immediately. In closed drainage the use of normal salt solution or Dakin's solution for irrigation, where there are thick fibrin clots, is very effective to help insure proper drainage.

There are a few specific facts that should be kept in mind in the surgical treatment of acute empyema whether closed or open type drainage is used. Do not establish wide open drainage until there is good evidence that the mediastinum is fixed. Any tube that is used for drainage should be anchored so there is no danger of its loss in the cavity. Inadequate or non-dependent drainage are the two most common causes in the development of chronic empyema. The morbidity and mortality rates will be cut to a minimum if proper, adequate drainage is established soon after a frank acute empyema has developed.

# Hemorrhage from Meckel's Diverticulum in Infancy

## Report of a Case

William B. Patterson, M.D.

Puunene, Maui

Meckel's diverticulum is estimated to occur in from 2 to 4 per cent of all infants, though it rarely causes any symptoms. At times, however, it does produce symptoms and gives rise to pathological conditions within the abdomen. The tip of a Meckel's diverticulum may become attached in the abdomen and produce intestinal obstruction. The diverticulum may become inflamed and produce the signs of acute appendicitis. It may invert itself into the lumen of the ileum and lead to intussusception. It may develop ulceration which may perforate and cause peritonitis. There may be massive hemorrhage from the ulceration which can be fatal if unchecked. All of these conditions except hemorrhage will be accompanied by signs and symptoms that will necessitate an exploratory abdominal operation.

Hemorrhage from the bowel may be the only symptom of a Meckel's diverticulum. Physical examination of a patient with a bleeding Meckel's diverticulum will be negative except for signs of anemia. Examination of the blood will show anemia but no other abnormality. X-ray of the gastro-intestinal tract is usually normal. The hemorrhage may show as a tarry stool, but if it is massive, and in an infant, it may produce a red, liquid stool. Sometimes blood clots will form in the stool when a large hemorrhage occurs. If there are repeated small hemorrhages a severe secondary anemia will develop. If the hemorrhage is large the patient may go into shock and die if not properly treated. The following case is recorded to illustrate the need for immediate transfusion and abdominal operation in infants with massive intestinal hemorrhage for which no cause can be found.

### Case Report

R. N., male, weighed 9 pounds 2 ounces at birth and appeared normal. His mother had syphilis but received adequate treatment during pregnancy. He attended monthly baby clinics and developed normally for the first eight months. At this time the mother reported that his stools were very dark in color. This was thought to be of dietary origin. The mother was told to bring him to the clinic if the dark stools continued.

All was well until eleven months of age, at which time he passed a large bloody stool. I saw the diaper he was wearing at the time and there was no doubt that the stool consisted almost entirely of blood. The mother stated that he had had a normal bowel movement twelve hours previously and had not been ill. The temperature was found to be 101 F. and the skin was pale and clammy. His pulse was very rapid. Otherwise the examination was negative. He was given 1/32 grain of morphine sulphate and 1 mg. of vitamin K hypodermically.

A blood count revealed 38 per cent hemoglobin and 2,700,000 red cells. The white blood count was 13,400 with 15 per cent neutrophils and 85 per cent lymphocytes. The coagulation time was two minutes and the bleeding time was one-half minute. The blood was type A. The urine was normal.

The patient was observed for twelve hours and was given a milk diet. During this time his condition grew worse, although he did not have another bowel movement. The respirations were thirty-four per minute and the skin was paler. He was given 165 cc. of whole blood in a scalp vein. His weight was 19½ pounds. After the transfusion he appeared much improved and his lips were of a better color. The next day his condition was improved and his temperature was normal though his hemoglobin was only 67 per cent of normal. He still had not had a bowel movement since just before admission. He was given another transfusion of 195 cc. of blood and just as this was finished he passed a large bloody stool estimated at 400 cc. During the next four days his condition was good and he had no bowel movement. On the fifth day after the last bloody stool he passed a normal stool with no gross blood in it.

The patient was seen from the onset of his illness by a surgical consultant. The possibility of a bleeding Meckel's diverticulum was discussed though it was deemed advisable to postpone operation until definite cause could be found for the bleeding. During the next three weeks there was only one bloody stool, though the hemoglobin did drop to 57 per cent of normal. During this period the baby was fine except for occasional cramp-like pains in the abdomen. Digital rectal examination was negative. X-rays of the entire gastro-intestinal tract were negative. The blood Wassermann reaction was negative. He took a regular diet for his age during this period. Finally, after three weeks, he was allowed to go home with the instruction to return immediately if any blood appeared in the stool. He was given a liquid medicine to take which contained liver extract and ferrous sulphate.

During the next three months he returned to the clinic at ten-day intervals for examination and hemoglobin determination. The hemoglobin gradually rose until it reached 75 per cent of normal. His general condition gradually improved, though he did not return to normal. He gained only one pound during the four months following the first bloody stool. He was unable to stand alone although he was over fifteen months of age. While at home no gross blood was ever seen in the stools though a few specimens of stool did contain a small amount of blood by laboratory test.

At fifteen and one-half months of age he passed another large bloody stool. Following this his hemoglobin dropped to 55 per cent of normal. He was prepared for operation by a transfusion of 100 cc. of blood and by four doses of 3¾ grains (0.25 grams) of sulfadiazine by mouth at four-hour intervals preceding the operation.

At operation, through a low midline incision, a Meckel's diverticulum was found at the usual location on the ileum. The ileum distal to the diverticulum, and the colon, were filled with blood. There was an adhesion near the base of the diverticulum. The diverticulum was removed and the intestine was closed with three layers of sutures. In the

fresh state the diverticulum showed a firm blood clot attached to the mucosa near the base.

Immediately after the operation the patient was given 250 cc. of blood. His condition was good and remained good. He was bothered by abdominal distention but this was relieved by suction through a Levine tube. He was given sulfadiazine post-operatively. His temperature fluctuated between 100 F. and 102 F., and after the third day he was given 5000 units of penicillin every three hours for three days. Following the institution of penicillin treatment his temperature dropped to normal and he made an uneventful recovery. He went home on the tenth post-operative day.

The tissue specimen was fixed in formalin and was sent to Dr. I. L. Tilden for study. He described it as a small structure, 3 cm. long and 1 cm. in diameter, with a lumen 5 mm. in diameter. Microscopically, the structure was lined by thickened bowel mucosa. There was evidence of chronic inflammation, with fibroblastic proliferation and collections of lymphocytes. He was unable to demonstrate either an ulcer or a bleeding point but stated that it was very difficult to demonstrate a bleeding point after formalin fixation. Diagnosis: Meckel's diverticulum with chronic inflammation.

After going home the patient continued to improve. In the three months following operation he gained three pounds of weight. He began walking at eighteen months of age and now appears normal though small for his age.

## SUMMARY

1. A case of massive hemorrhage from a Meckel's diverticulum in an infant is reported. The first hemorrhage apparently occurred at eight months of age.

2. The only symptom and sign of a Meckel's diverticulum may be profuse hemorrhage from the bowel and secondary anemia.

3. The only treatment for hemorrhage from a Meckel's diverticulum is prompt transfusion of whole blood and surgical removal of the diverticulum.

4. In the surgical removal of a Meckel's diverticulum, the incision should be made transverse to the lumen of the ileum and it should be closed transversely to prevent the later development of intestinal obstruction. Care should be taken not to remove any of the ileum.

5. Delay in the removal of a Meckel's diverticulum after it begins to bleed leads to chronic secondary anemia which retards growth and development.

# HAWAII MEDICAL JOURNAL



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# EDITORIALS

## THOUGHTS OF A DOCTOR WHILE AWAITING RED TAPE UNWINDING IN THE LOCAL O.P.A. TIRE RATIONING OFFICE

He had rushed through house calls the previous morning in order to have his badly worn tires inspected and approved for replacement.

Between hospital rounds and office hours he had sandwiched in a visit to the O.P.A. office to arrange for the tire replacements.

He had arrived there at 8:45 A.M., only to discover that the O.P.A. office now opened up for business at 9:00. (Outside of government employees, most people he knew started the day's work by 8:00 or at least by 8:30; he had started at 6:30.) So he sat on the steps and waited, filling out a long and tedious questionnaire as he did so.

Then—although the inspector's report indicated his tires were dangerously worn—he was told no one in the office was qualified to pass on the application, but the Board (fortunately) was meeting that day and would consider the matter. He could return the next day for the necessary document which would permit a dealer to sell him tires. No, the office could not (or would not?) mail this to him; the doctor must make another trip ("was this trip necessary?") next day to pick it up.

So next morning—this was the third day—he postponed a house call on a sick child to revisit the tire rationing office. He could have saved time and mileage by stopping in on his way from one hospital to another had the office been open when most private business offices open; but it wasn't.

One of the office employees leisurely rose from her desk, took a final drag on her cigarette, put out the fire, snapped the butt through an open window, then asked what the doctor wanted.

She wasn't the lady he had talked with the day before, and expressed some astonishment when he said he had filed his application the previous day and expected a permit to buy tires today. "Why, our Board just *met* yesterday after*noon*!" Then, "Oh, are you a doctor? Wait a minute."

So he sat, while a couple of other girls rummaged through a foot-high stack of forms, questionnaires, and God-knows-what. (Just how did anything ever get done back in the good old days before bureaus and bureaucrats developed their technic for producing forms and questionnaires?)

And the doctor sat on a wooden bench and waited, and while he waited he mulled over in his mind the compulsory health insurance scheme which is part of the Wagner-Murray-Dingell bill now pending in Congress, and pondered on its implications.

With the working of an office of a government bureau unfolding before his nose he couldn't help comparing two processes: (1) how he used to buy tires, and (2) how he bought them now. He used to call up a dealer of his choice and tell him he needed new tires. The dealer, glad to have his business, sent for the car, put on the tires, and sent in his bill; it was paid, and that was that. Now—well, the government had moved in between. With the rubber shortage, there was reason and need for it, to be sure. But *when* the government moved in, a simple process which used to take an hour or so now required three days, three two-mile auto trips, endless forms to fill out, the action of a board, and the functions of an office staffed with "servants of the people" observing bankers' hours.

The two processes, the old and the new—or, in regard to sickness, the present method of getting taken care of and the one proposed—came back to mind. Now, if someone is sick, he goes to a doctor and the doctor is responsible to him. If the doctor doesn't give him good service, or fails to help him, he goes

elsewhere. That's an incentive for the doctor to give all he's got, or he won't have any practice. And it is still a good old-fashioned custom in most communities for the doctors to take care of the sick first, and take up the matter of payment later.

How will a person get medical care under the Wagner-Murray-Dingell act? Senator Wagner says everything will be just as it was. BUT THE GOVERNMENT WILL BE IN BETWEEN! Just as the ration board is between the doctor and his tires—and let no one think for a moment that that won't include all the delays, red tape, questionnaires and petty interferences that are part and parcel of about every governmental bureau one can name.

What about the doctor's responsibility? Today it is to the patient, directly and absolutely. Under Senator Wagner's bill, the doctor's responsibility will be to the government, to whom he will look for his pay. He will do as "the government"—meaning any or all of the little people who have the say-so in a government office—says. His chance of advancement in his profession, his very livelihood, will depend on his playing ball with the political or bureaucratic powers-that-be. Maybe that's all as it should be—but will the patient be better or worse cared for under such a system?

Selling a political ideology to the masses under the old malarky that they are going to get something for nothing seems to be the order of the day.

There are even handouts to the doctors themselves in Mr. Wagner's bill, and some doctors will undoubtedly fall for them.

Beware the politicians bearing gifts!

Look all the way down a Trojan gift horse's gastrointestinal tract!

There are ways of correcting the at present imperfect distribution of medical care, other than turning it over to the bureaucrats to administer.

The people have had an opportunity to learn a lot about government intervention in their private lives during this war. When the needs imposed by the war are gone, will they forget the lessons they've been taught, and have to learn them all over again the hard way?

P.S.: The doctor got his tires, but was an hour late to his office, and were the patients mad!!

## NO CHAGAS' DISEASE IN HAWAII

To the Editor:

Enclosed, for your information, is a report which Dr. David Bonnet, the entomologist who is employed by the health department, has made on the question of Chagas' Disease in Hawaii, after considerable study of this matter.

It seems to me that Dr. Bonnet's conclusions that the presence of Trypanosoma cruzi here is unproven and extremely doubtful, are well founded. We should not, however, completely close our minds to the possibility.

C. L. WILBAR, JR., M.D.,
President, Board of Health

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TERRITORY OF HAWAII
BOARD OF HEALTH
DENGUE MOSQUITO CONTROL

August 20, 1945

TO: President, Board of Health
FROM: Entomologist, Dengue Mosquito Control
SUBJECT: The Question of Chagas' Disease in Hawaii

1. Recently, considerable discussion and speculation has centered around the verbal report of Lt. S. F. Wood, Entomologist, 4th Marine Division, that he had found *Trypanosoma cruzi* in the local species of Reduviid, *Triatoma rubrofasciata.*

2. Dr. Walter Carter, Entomologist for the Pineapple Experiment Station, reported at the July meeting of the Hawaiian Entomological Society for Lt. S. F. Wood that this trypanosome had been identified as *Trypanosoma cruzi* and that Dr. H. Arnold, Jr., and Dr. D. Bell had previously reported cases now believed to be Chagas' disease. (HAWAII MED. J. Vol. 3, Jan.-Feb., 1944.)

3. An intensive search was made for specimens of *Triatoma rubrofasciata* by myself and Bernard Brookman, P. A. Sanitarian (R) USPHS, and none were discovered. However, Mr. K. Sakimura of the Pineapple Research Station sent to me two nymphal forms. This species has been found principally in the Kaimuki district, but it has been also reported from Manoa and Nuuanu Valleys. I have been informed by Mr. E. C. Zimmerman, Entomologist of the Bishop Museum, that *Triatoma rubrofasciata,* an Oriental species of tropicopolitan distribution, has been found only on the island of Oahu in the Territory of Hawaii.

4. At the August meeting of the Hawaiian Entomological Society (August 13, 1945), Dr. Walter Carter presented for Lt. Wood a paper on this subject to be published in the proceedings of the Hawaiian Entomological Society for 1945. In this paper the trypanosome found in the gut of

*Triatoma rubrofasciata* from Hawaii is identified as *Trypanosoma conorhini* (Donovan). This trypanosome was originally described by Donovan (1909) from specimens of *Triatoma rubrofasciata* in India. It has since been reported harbored by this species of Triatoma in Java, Formosa and Mauritius. *Trypanosoma conorhini* is non-pathogenic, at least to rats and mice. The vertebrate host has been reported to be the house rat.

5. Summary and Conclusions.

(a) *Triatoma rubrofasciata* is found only on the island of Oahu in the Territory of Hawaii and in rather limited numbers. It is principally known from the Kaimuki District of Honolulu. This species is a cosmopolitan species and was probably introduced into Hawaii from the Orient. It is difficult to understand how a species of Oriental origin could become infected with the South American trypanosome, *Trypanosoma cruzi. Trypanosoma conorhini,* which has been described from this species of Triatoma in Formosa, Mauritius, and India, is very similar in appearance to *Trypanosoma cruzi* but possesses no leishmania tissue phase in mice, rats, and guinea pigs and has an extremely short vertebrate host cycle in the rat.

(b) *Trypanosoma cruzi* has not been found in the Territory of Hawaii; and hence, the presence of Chagas' disease, which is caused by this protozoan, is unproven and extremely doubtful.

Respectfully submitted,

DAVID D. BONNET
Asst. Sanitarian (R) USPHS
Entomologist, Dengue Mosquito Control

## VENEREAL DISEASE CONTROL: AN INTERIM REPORT

The following table gives the numbers of cases of gonorrhea, and primary and secondary syphilis, reported to the Hawaii Territorial Board of Health from Army, Navy and civilian agencies as having been acquired on Oahu during each of the months indicated. The periods covered are the nine months preceding, and the nine months following, the closing of the houses of prostitution on September 21, 1944.

| MONTH | GONORRHEA | SYPHILIS | MONTH | GONORRHEA | SYPHILIS |
|---|---|---|---|---|---|
| Jan. | 90 | 1 | Oct. | 88 | 3 |
| Feb. | 91 | 5 | Nov. | 74 | 5 |
| Mar. | 93 | 3 | Dec. | 53 | 4 |
| Apr. | 74 | 2 | Jan. | 68 | 3 |
| May | 120 | 13 | Feb. | 57 | 3 |
| June | 75 | 5 | Mar. | 43 | 5 |
| July | 65 | 7 | Apr. | 31 | 1 |
| Aug. | 116 | 4 | May | 53 | 3 |
| Sept. | 115 | 10 | June | 50 | 1 |
| TOTAL .. | 839 | 50 | TOTAL .. | 517 | 28 |

## ALOHA, CAPTAIN PLEADWELL

As this issue goes to press, Dr. Frank Pleadwell, Captain (Medical Corps), United States Navy (Retired), is leaving Hawaii for an extended Mainland trip with Mrs. Pleadwell. He expects to return to the Territory, but does not yet know how soon this will be.

Dr. Pleadwell has been an active and interested member of the Editorial Advisory Board of the HAWAII MEDICAL JOURNAL since the Board was established in September, 1943.

Dr. Hastings H. Walker, Director of Leahi Hospital, will take Dr. Pleadwell's place on the staff of the JOURNAL.

# COUNTY SOCIETY REPORTS

## HAWAII COUNTY MEDICAL SOCIETY

The 240th regular monthly meeting was called to order by Dr. R. Eklund at 7:15 P.M. on July 5, 1945, in the Hilo Memorial Hospital Staff Room. Fifteen members and 11 guests were present. The minutes were accepted and filed.

The secretary read a communication from the executive secretary of the Territorial Medical Association, which answered our query regarding the status of the medical library in the Mabel Smyth Building. This is a Honolulu County Medical Library and receives its chief support from the donation of fees paid to Honolulu County doctors for treatment of indigent cases. The letter suggested that the councillors might bring up at the next annual territorial meeting the question of dividing the annual contribution from the Territorial Association among the active county medical libraries.

Two movies were shown—*Human Sterility,* sponsored by the local Board of Health, and *Vitamin Deficiency,* presented by Squibb & Sons.

Since this was his last meeting before departure for his new assignment on the Island of Molokai, our president, Dr. Eklund, bade farewell to the society and Dr. Leslie, vice president, assumed the chair. Dr. Leslie announced a special meeting to be held in honor of Dr. Eklund on July 7 at Hilo Country Club.

The meeting adjourned at 9:20 P.M.

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A special dinner meeting was held at the Hilo Country Club at 7:30 P.M. on July 7, 1945. Eighteen members and 4 guests were present to wish good luck to our former president, Dr. R. Eklund.

Three movies were presented by the local Board of Health. Dr. Yoshina led the discussion following the first movie, which was on differential diagnosis and therapy of vomiting in the newborn. The second picture was on eye surgery and Dr. Crawford commented on it. Dr. Yoshina also discussed the third film on the subject of laryngeal obstruction.

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The 241st regular monthly meeting of the Hawaii County Medical Society was held in the Hilo Memorial Hospital Staff Room on August 2, 1945. Dr. W. Leslie called the meeting to order at 7:40 P.M. Seventeen members and guests were present. The minutes were placed on file.

An invitation to a dinner meeting for Capt. Borst, Commanding Officer of Corps Evacuation Hospital No. 1 was extended to the society by Commanders Hale and Sterner. It was decided that this meeting should be considered as the regular September semi-annual medical meeting of the society.

Mr. Reginald Carter of the Hawaii Medical Service Association outlined the work of that organization and the plans for establishing a similar association in Hilo.

It was moved that this society go on record in favor of such a plan for the Island of Hawaii and sponsor the proposed association with the assistance of Mr. Carter. The president appointed Drs. Sexton, Crawford and Orenstein a committee to sponsor such an association. Mr. Carter and three local business men will be added to the committee. Mr. Carter also asked that the present officers of the Hawaii County Medical Society serve on the committee for the time being.

Dr. Larsen announced that a definite plan for a skin clinic, sponsored by the Plantation Physicians' Association, is now in the hands of the managers. It is planned that Dr. Johnson, the consultant, will make regular clinical visits to this island, during which a day will be allowed for private consultations.

Meeting adjourned at 9:30 P.M.

S. MIZUIRE, M.D.,
*Secretary*

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## MAUI COUNTY MEDICAL SOCIETY

The regular meeting of the Maui County Medical Society was held August 21, 1945.

Members present were Drs. Von Asch (presiding), K. Izumi, Patterson, Kushi, Kanda, Rothrock, Osmers, T. P. Chou, Ellen Chou, McArthur and Sanders. A large number of Marine and Navy doctors came as guests.

It was decided that fees for the pre-marital examinations required by law should be determined by the individual examiners, based on the extent of the examination. No fee is allowed to government physicians for making such examinations.

Two sound films were shown, one on *Modern Nutrition,* the second on *External Skeletal Fixation of Fractures of Tibia and Os Calcis.* The latter was presented by a Navy doctor, who also gave a paper and led a discussion on the same subject.

JOHN SANDERS, M.D.,
*Secretary*

# NEUROPSYCHIATRIC COMMENT

## CIRCUMSCRIBED NEURODERMATITIS

Circumscribed neurodermatitis is one of the commoner dermatologic problems, and one of the most difficult to manage. It can seldom be cured by treatment of the skin alone, and many patients consequently suffer from it for years in spite of repeated visits to many doctors. It can often be controlled, however, and occasionally cured, if the physician recognizes it and bases his treatment on rational grounds.

### The Neurodermatitides

"Neurodermatitis" is an ill-defined term which means various things to various men. It implies in general a pruritic dermatosis based on non-organic dysfunction of the central nervous system. In this country, it is usually divided into three main categories: disseminated neurodermatitis, acute exudative neurodermatitis, and circumscribed neurodermatitis.

*Disseminated neurodermatitis* is used in some schools as a synonym for atopic (flexural or infantile) eczema, and in some as a synonym for multipie and widespread lesions of circumscribed neurodermatitis. It has not, in general, been sufficiently clearly defined to be a very useful term.

*Acute exudative neurodermatitis* is an acute and severe, often widespread, eczematous disorder, which would require a monograph for its description: it is most often diagnosed by exclusion of the first-suspected physical or chemical causes. It is clearly described and well illustrated in Becker and Obermayer's *Modern Dermatology and Syphilology.*

*Circumscribed neurodermatitis* is by far the commonest of the three varieties of the disease; it has gone by various names—eczema papulosum, lichen simplex, lichen Vidal, lichen simplex chronicus, pruritus, pruritus vulvae, and pruritus ani.

### Diagnosis of Circumscribed Neurodermatitis

*Paroxysmal pruritus* is the most valuable diagnostic feature of the disease. It is almost invariably the chief complaint, and it is rarely met with in any other disease. The paroxysmal feature of it is the important thing; and by this is meant three things: the itching is (1) *severe,* (2) *intermittent,* and (3) usually *abrupt in onset.* It is almost always so severe that the patient is compelled to scratch or rub the itchy place, often violently; it is entirely absent for periods of several hours at a time, especially when the patient is pleasurably occupied; and the onset of paroxysms is frequently so abrupt that it actually makes the patient jump. The patient will often confess, sometimes sheepishly, that there is a lot of pleasure obtained by scratching, and a sensation of relaxation and relief afterward. The analogy with the sexual orgasm is obvious.

*Circumscribed lichenification* is the second most characteristic feature of the disease; it is so characteristic that, like the pruritus, it may actually be diagnostic. The lesion usually has a clearly demarcated edge, which is frequently slightly elevated and is likely to be smoothly curved, forming an oval plaque. This plaque is typically lichenified, that is, thickened in a leathery fashion, with its surface cross-hatched by the rhomboid pattern of the interfollicular lines of the skin. The depth of these lines is exaggerated. There are often punctate excoriations, especially in early cases. Increased pigmentation is common.

*Site of involvement* is highly variable, but the sides or back of the neck; the shin; the forearm; the elbow, knee, or ankle flexures; and the genital and perianal regions, are much more often involved than all other areas together. The face, scalp, palms and soles, and trunk are rarely involved.

*Histologic changes* may be useful, though they are rarely necessary, in excluding alternative diagnoses. The histologic changes are not absolutely diagnostic. They most closely resemble those of psoriasis: rather uniform acanthosis (thickening of the epidermis with regular, orderly elongation of the rete pegs) and hyperkeratosis, often with parakeratosis. There is a moderate to marked round-cell infiltration of the corium, usually with more eosinophiles than one finds in psoriasis.

### Etiology

Although the *Standard Classified Nomenclature* still lists this disorder under "diseases of unknown or uncertain etiology," it seems fairly clear that many if not most cases of it are an expression of emotional tension or conflict. Stokes has discussed this aspect of the problem fully and well, and his paper (*J.A.M.A.* 105:1007, 1935) should be read for a more nearly adequate understanding of the subject. The monotonous regularity of failure of topical applications alone, and of relapse after x-ray therapy, makes it fairly plain that there is much more to the disease than meets the eye. The frequency with which the disease is observed in tense, high-strung, high-pressure peo-

ple, people with a high standard of behavior, or people on whom heavy demands for performance or adjustment are being made, is striking, and leaves little room for doubt that the disorder is not merely somatic but pre-eminently psycho-somatic. It is as if the pruritus were a safety valve with which to release excess emotional steam; or, to put it in another way, as if the skin were serving as whipping-boy for an emotional problem—the patient gratifies his urge to solve the problem, by scratching the itch instead. As previously mentioned, the parallel with the sexual orgasm is a close one, so close, indeed, that patients not infrequently are visibly embarrassed by their realization of the similarity. The frequency of involvement of the genital and anal regions is probably significant in this regard, as is the frequency with which unsatisfactory (or lacking) sexual relationships are discovered as potential or actual causative factors.

### Treatment

Treatment falls under two headings: topical symptomatic measures, and psychiatric measures. It is important to realize, *and to convince the patient,* that *local treatment is not likely to effect a cure.* To be sure, it occasionally does effect a cure, but this is unusual and should not be relied upon. This conviction can usually be imparted to the patient simply by explaining the etiology of the disease.

The aim of topical treatment is symptomatic relief. Three kinds of topical measures have value: (1) antipruritic and keratolytic preparations (crude coal tar paste or varnish is one of the best; White's formula, or 5 per cent crude coal tar in chloroform, is suggested); (2) x-ray therapy (75 or 100 roentgens unfiltered or lightly filtered, once a week, or double this dose once every two weeks, for a month or two, may be tried), or (3) vigorous maceration of the lesions every two or three weeks with a stout gauze swab wet with 20 per cent potassium hydroxide solution, until the skin is sore, following which it is mopped with boric acid solution, blotted dry, and dressed daily with boric acid ointment until it is healed. This last method is not suitable for anogenital lesions.

Psychiatric treatment is usually required for permanent relief. It involves principally discovering, by questioning the patient, situational factors which may lead to emotional conflict and tension. It may be possible for the general practitioner or dermatologist to accomplish this in his office, with an intelligent patient and adequate skill and good luck; however, the patient is often unconscious of any etiologic factors, and reference to a psychiatrist may be required. It is difficult to know how many cures are accomplished by psychiatric treatment, but it is not at all difficult to know how many lives of "quiet desperation" are brought to the attention of the psychiatrist by reference of these cases. It is very rare for such a reference to "draw a blank," so to speak.

### Prognosis

The prognosis for these cases by the all-too-common practice of going from doctor to doctor in search of the "right" topical application is clearly exemplified by the average victim of pruritus ani or pruritus vulvae. It is very poor indeed. Chronicity is the almost invariable rule.

Follow-ups are difficult, and seldom obtained, and so the prognosis *with* psychiatric management has not been established; but the prompt relief observed in the occasional case suggests that success is at least as frequent as failure. At all events this approach to these cases is a constructive one, calculated to help them generally; whereas the old hit-or-miss method of searching for the proper ointment or lotion is fairly certain to be a failure, and even x-ray therapy is so uncertain, and so likely to eventuate in x-ray dermatitis, that it cannot be recommended except for the occasional desperate—or very elderly—patient.

HARRY L. ARNOLD, JR., M.D.

The Clinic, 881 So. Hotel St.

# HOSPITAL NEEDS

## THE CONVALESCENT-NURSING HOME

In the January-February, 1944, issue of the JOURNAL an article was written under the caption, "The Question of a Convalescent-Nursing Home." Today, though the answer is not yet an actuality, there is no longer any question.

The analysis made by the County Medical Society in the spring of 1943 established the need. In the intervening months there have been discouragements and postponements and bogged-downedness; a flare-up, a dying down; a blowing hot, a cooling off; property within grasp, property withdrawn; and over all the tensions consequent to the War; but the flame never quite went out, and at the moment it is burning brightly. As was to be expected, the War was our greatest deterrent to carrying through at an earlier time. Not only were we aware of strains and stresses, but labor and material were non-existent. Looking back, we can see that the very difficulties we encountered were but strengtheners; fighting resistance carried us through.

For one thing, in the beginning the opinion concerning the need of a convalescent-nursing home was not a unanimous one; even the doctors were not altogether in accord, as they seem to have since become; more time was required for the idea to soak in and develop. All the while three questions persistently came to us in one form or another, came from doctors, patients, families and the community at large; nor were there any answers: where can a patient go who needs long convalescence but who no longer needs hospitalization? The residence of such a patient may be a hotel; even if he lives in his own home, there may be no one in it who can give him the required care. He is perhaps getting depressed, or too dependent on the hospital, and he needs, therefore, a more stimulating environment to bring him back to normal. Where can one stricken in the prime of life get adequate care? He may live many years but as a semi-invalid. And finally the third question: where can elderly people have the kind of attention they require? Some of them are occupying hospital beds needed by those who are acutely ill, some are living alone in hotels, or in their own homes or with their families, but the families have come to feel the strain is too wearing, especially where there are young folk under the same roof.

Many a time when the light was burning low, nigh to going out, when it seemed as though the obstacles were too many and we were on the verge of giving up, a doctor would call or a patient grow anxious or a tired relative would plead, and we would spark again. And so through all the vicissitudes since the spring of 1943, we have kept the light burning even though at times it was no more than a flicker.

The enforced waiting has made for unanimity of opinion, throughout the community. For months we haven't, as we did in earlier days, met with negative ideas — everywhere and on all sides we find approval: doctors, business leaders, organizations, churches, the man in the street and the press are unanimous now in their belief that a convalescent-nursing home should be established.

In the early days of our endeavors we thought the first essential was a site, the second incorporation. We have reversed the order and on September 24, 1945, under the laws of the Territory, the Convalescent-Nursing Home was incorporated. The following were the incorporators: Messrs. Riley H. Allen, Stafford L. Austin, Raymond S. Coll, Sr., Cyril F. Damon, Alan S. Davis, Peter K. McLean, George H. Moody, C. T. Oliphant, C. Dudley Pratt, Alva E. Steadman, Miss Mary Catton and Dr. F. J. Pinkerton.

As a charter and by-laws require directors, these same persons will so act. Later, when the home is established, there will be some change in the directorate. It calls for a minimum of 9 and a maximum of 19 persons. Since September 24, the following have also been added as directors: Bishop H. S. Kennedy, Bishop Jas. J. Sweeney, Reverend Allen Hackett, Mr. Ralph E. Woolley, Mr. Henry Inn, Mrs. Mark Robinson and Mr. Scott Brainard.

Incorporation has given legal sanction to the idea of a convalescent-nursing home, and makes possible the acceptance of funds against the time we can actually acquire property. At the moment we have no site, and the name, "Convalescent-Nursing Home" is merely an expedient for corporeity. We think that later we will be able to find a more pleasing name, perhaps one suggested by the very site that is chosen for the Home.

We think in terms of 100 patients, or boarders, to begin with, and of sufficient acreage, not only in the interest of normal expansion but to make possible a division between age groups as well as between

convalescents and the chronically ill. We envision a home that will breathe beauty and peace and comfort. Attention will be given to suitable programs. The younger patients and convalescents will be encouraged to participate in activities suitable to their particular conditions, but sufficiently active to stimulate independence and a desire for normal health, emotional as well as physical. For the aged there will be a more passive regimen in a quieter part of the Home, one which will offer suitable diversion but most of all will give them a sense of security and loving care to the end of the road.

Though nothing has been decided in this regard, some of us envision a large central administration building where those needing most attention will live, and in landscaped grounds there will be units for those needing less attention or for aged couples, financially independent, but who no longer want the worry of maintaining their own larger homes—worry of servants, taxes, repairs, etc. Such couples may build and equip cottages approved by the directors, which will give them the privacy of a separate home without the burden of its upkeep. After they have gone, the units will revert to the Home.

The Home will not be under the control of any vested interests; it will have its own board of directors. Because it will not have the heavy overhead of a general hospital, X-rays, surgery, laboratory, etc., nor its large numbers of trained personnel, the cost for accommodation, whether ward or private room, will not by any means be as high as in a hospital equipped to care for the acutely ill. It will be non-racial, non-sectarian and Territory-wide. Patients may, as they do in their own homes, call the doctors of their choice when medical attention is required.

Patients or boarders in the Convalescent-Nursing Home will get the nursing and dietary care their particular states require. If they develop conditions needing surgery or such professional services as hospitals furnish, their doctors may have them transferred to a hospital for as long as is necessary.

As the home crystallizes, policies for admission and management will be worked out. We mean it to be well managed and adequately supported. It is felt that to get the Home we want we will need to raise at least $350,000. The idea is to write off the original capital outlay investment, thus making the costs to patients lower than they would be otherwise.

The drive for funds will have started by the time this goes to press. The able chairman is Henry A. White, with Philip E. Spalding, Jr., and Calvin S. White as co-chairmen. There is no deadline for the campaign, though we hope to have ample funds by the end of the year to make a beginning. Doctors have a peculiar opportunity, and may we say an obligation, for helping toward the realization of a Convalescent-Nursing Home for Hawaii; we hope they will talk about it as they go in and out among their patients, that they will let it be known that contributions may be made as cash donations, as memorials, as expressions of gratitude or as bequests in wills, that gifts may be made in money or kind, may be given in cash or in the form of building a unit or furnishing a room. This will not be difficult, for there is one unique factor which differentiates this campaign from any other hitherto, and that is that everyone now has, or will have some day, some kin or friend who is either in need of convalescent care or who, chronically ill or aged, is in need of nursing care.

Checks should be made out to the Convalescent-Nursing Home, and addressed to P.O. Box 3474, Honolulu.

MARGARET M. L. CATTON

# LIBRARY NOTES

## THE HONOLULU COUNTY MEDICAL LIBRARY

MRS. ETHEL HILL, Librarian

MISS DORIS T. YASUTAKE, Library Assistant

8:00 A.M.–4:30 P.M. Phone 65370 7:30 P.M.–9:30 P.M.

### RECENT ACQUISITIONS

By Purchase:

Brock, Samuel: *The basis of clinical neurology.* 2nd ed. c1945.
Manson Bahr, Sir Philip: *The dysenteric disorders.* 2nd ed. c1942.
Moorhead, J. J.: *Clinical traumatic surgery.* c1945.
Rich, A. R.: *The pathogenesis of tuberculosis.* c1944.
Rogers, Sir Leonard: *Tropical medicine.* 5th ed. c1944.
Stroud, W. D.: *The diagnosis and treatment of cardiovascular disease.* 2v. c1945.

From the NURSES' ASSOCIATION

Anderson, G. W.: *Communicable disease control.* c1941.
Beck, A. K.: *Reference handbook for nurses.* c1941.
Cooke, W. R.: *Essentials of gynecology.* c1943.
Davis, M. E.: *DeLee's obstetrics for nurses.* c1944.
Eliason, E. L.: *Practical bandaging.* c1938.
Emerson, C. P.: *Essentials of medicine.* c1940.
Flikke, J. O.: *Nurses in action.* c1943.
Gilbert, Ruth: *The public health nurse and her patient.* c1940.
Gladwin, M. E.: *Ethics, a textbook for nurses.* c1937.
Greenhill, J. P.: *Office gynecology.* c1943.
Grinker, R. R.: *Neurology.* 3rd ed. c1943.
Hawley, Gertrude: *The kinesiology of corrective exercise.* c1937.
Hess, J. H.: *The premature infant.* c1941.
Hume, E. E.: *Victories of army medicine.* c1943.
Lyon, R. A.: *Mitchell's pediatrics and pediatric nursing.* c1944.
McBride, E. D.: *Crippled children.* c1937.
O'Hara, F. J.: *Psychology and the nurse.* c1943.
Pennock, M. R.: *Makers of nursing history.* c1940.
Rand, Winifred: *Essentials of pediatrics.* c1936.
Ribble, M. A.: *The rights of infants.* c1943.
Robinson, G. C.: *The patient as a person.* c1939.
Robinson, Victor: *The story of medicine.* c1943.
Rue, C. B.: *The public health nurse in the community.* c1944.
Sewall, Mary: *Trends in nursing history.* c1944.
Sellew, Gladys: *The nursing of children.* c1942.
Sellew, Gladys: *Sociology and social problems in nursing service.* c1941.
*State board questions and answers for nurses.* 23rd ed. 1945.
Steele, K. M.: *Psychiatric nursing.* c1943.
Taylor, A. M.: *Ward teaching.* c194..
Williams, B. C.: *Clara Barton, daughter of destiny.* c1941.
Zabriskie, Louise: *Nurses' handbook of obstetrics.* c1943.

From the AMERICAN MEDICAL ASSOCIATION

*New and non-official remedies,* 1945.

From the UNIVERSITY OF SYDNEY, SCHOOL OF PUBLIC HEALTH AND TROPICAL MEDICINE

*Collected papers.* #1, 1937.

By Gift:

Grinker, R. R.: *Men under stress.* c1945.
Maisel, A. Q.: *Miracles of military medicine.* c1945.

### JOURNALS CURRENTLY RECEIVED

The following journals are being *currently* received in the Library. Please add them to the list previously published in the November-December 1944 issue.

Annals of Tropical Medicine and Parasitology
Journal of Tropical Medicine and Hygiene
Medical Journal of Australia

### JOURNALS NEEDED

With the addition of journals from the Medical Group and The Queen's Hospital internes, a large box of medical journals was packed and turned over to the Red Cross for immediate shipment to the Philippines. The Medical Library is unable to supply all issues of other journals that are also needed. If any doctor has copies of the following journals for disposal, please call Mrs. Hill at the Library:

American Journal of the Medical Sciences. 1942.
American Journal of Public Health. 1942–1943.
Archives of Internal Medicine. 1942–1943.
Surgery Gynecology and Obstetrics. 1942–1944.

While MRS. ETHEL HILL, our medical librarian, was on leave of absence on the west coast, she visited other medical libraries in Los Angeles, San Francisco, and Spokane and made many interesting contacts. She reports that outside of our present lack of space for development of journal files, the Honolulu County Medical Library is the equal of, and is in some ways superior to, any other library of comparable size which she visited.

## BOOK REVIEWS

*Common Ailments of Man.* Edited by Morris Fishbein, M.D. Cloth. Price, $1.00. Pp. 177. Garden City Publishing Co., 1945.

This book is a collection of sixteen articles dealing with common disease entities or symptom complexes in language understandable by the layman. Each article is by a recognized authority in the field concerned. All have been published previously in *Hygeia.*

Sample titles are "Backache," by Frank Ober; "Anemia," by William Dameshek; "Heart Disease," by Paul White.

The book can be recommended as authoritative, modern, understandable, and a useful reference for the layman of average intelligence. It might also be useful for the doctor who has difficulty finding adequate substitutes for polysyllabic technical words for explaining patients' diseases to them.

*How to Make a Speech and Enjoy It.* By Helen Partridge. Paper. Price, 75c. Pp. 24. New York: National Publicity Council, 1944.

This is a useful, practical, sympathetic manual of practice for the amateur public speaker. It tells him what to do, step by step, from the time he agrees to make a speech till the time it is over. The approach is a constructive one, with more do's than don't's. The book can be recommended unreservedly for the inexperienced orator.

*My Second Life: an Autobiography.* By Thomas Hall Shastid, M.D. Cloth. Price, $10.00. Pp. 1174. Ann Arbor: George Wahr, 1945.

A liberally illustrated and rather discursive second book on this subject, by the same author.


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Ampules, 0.5 cc.: Boxes of 6, 50, 100.
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*Literature on Request*

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# NOTES AND NEWS

## PERSONALS

DR. JOHN MILNOR, son of DR. AND MRS. GUY C. MILNOR of Alewa Drive, returned in September from Temple University to intern at The Queen's Hospital. DR. ELIZABETH JENNIE SMITH from the University of Iowa became the third woman physician now interning at Queen's. In October three more doctors arrived: DR. JAMES WILLIAM CHERRY of the University of Arkansas, DR. CASIMIR ANTHONY DOMZALSKI and DR. GRANT MINER WEDGE from the University of Michigan.

A surgical residency has been added to the existing residencies in medicine and neuropsychiatry at The Queen's Hospital, and acceptance by the A. M. A. is now under way. DR. JOHN CHALMERS, a St. Louis College graduate before further studies at Northwestern University, will be the first incumbent, beginning October 15.

LT. JAMES WONG, an Olaa resident who attended the University of Hawaii before taking his medical work at Jefferson Medical School, and a veteran of the Okinawa campaign, has been dining in Manila lately, where two servants are employed by his hosts only to keep the flies off the food and the guests.

DR. AND MRS. GARDNER BLACK, DR. AND MRS. SAMUEL WISHIK and DR. AND MRS. H. L. ARNOLD, SR., have recently returned from the mainland. DR. AND MRS. FRED LAM left for a trip to the states August 31. DR. CLIFFORD KOBAYASHI is maintaining Dr. Lam's practice during his absence.

Hawaii's public health nurses are the second best trained group of nurses in the United States and its territories, according to a report of the Federal Security Agency of the U. S. Public Health service. Oregon ranked first, and Alaska third. In January 1945 Hawaii had eighty-seven public health nurses, sixty-five of whom had public health nursing training. Registered nurses may take the public health nursing course at the University of Hawaii for one year and receive a public health nursing certificate.

MISS MARGARET P. EATON joined the Occupational Therapy Staff of The Queen's Hospital in July, replacing MISS JEAN TOMLINSON, who returned to the mainland in mid-September.

MISS BARBARA ANN BROWN, daughter of DR. AND MRS. S. R. BROWN of Hilo, was married to LT. (JG) JAMES DANIEL LANDON, USNR, son of CHIEF AND MRS. FRANK J. LANDON of Tucson, Arizona, on July 20 in Tucson.

LT. COL. J. E. WALTHER of Kauai was married recently on the mainland.

DR. FORREST J. PINKERTON was married to Mrs. Florence Helmick Macaulay on September 14, 1945. The wedding took place in Central Union Church, Honolulu.

Miss Cherry Young, daughter of Mr. and Mrs. Young Goon, became the bride of DR. THOMAS CHANG on September 1 in Wailuku. DR. CHANG is resident pathologist of The Queen's Hospital.

CAPT. DONALD S. DEPP has reported his new address; Dibble General Hospital, Menlo Park, California.

DR. PAULINE G. STITT, assistant director of the bureaus of maternal and child health and crippled children of the Board of Health, has resigned and left for the mainland, to become regional medical consultant of the Children's Bureau of the department of labor for the southeastern section of the United States. DR. STITT has been with the health department here for two years.

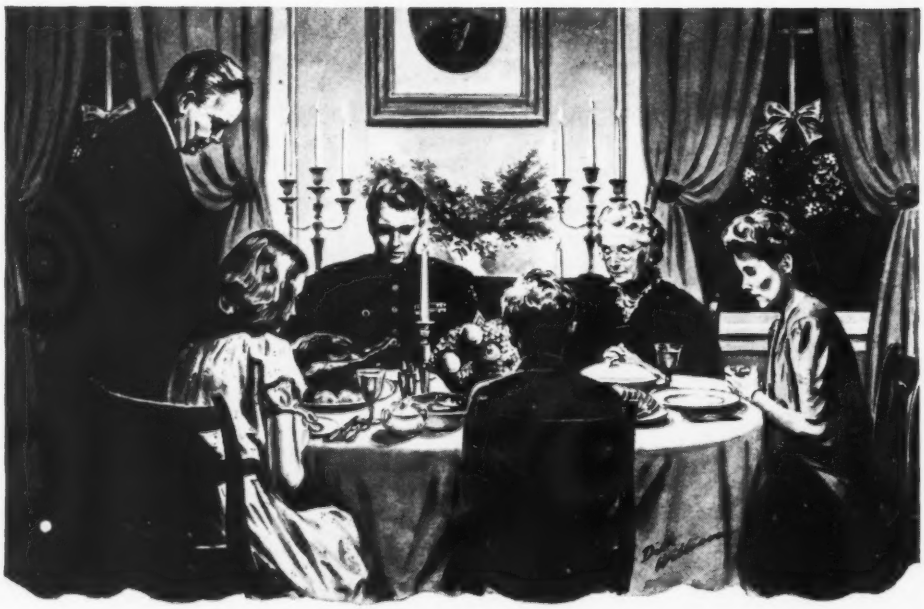
DR. BERNARD WITLIN, acting director of the bacteriological laboratories of the Board of Health, left recently for the mainland under orders to join the Ohio State Health Department.

DR. R. T. EKLUND has moved from Pahala, Hawaii, to Molokai, where he is now practicing. DR. WILLIAM LESLIE has succeeded him as President of the Hawaii County Medical Society. DR. LESLIE KASHIWA is the new physician at Pahala.

MISS PATIENCE L. CLARKE recently returned from the mainland to organize and administer a program of tuberculosis nursing education at Leahi Hospital.

This teaching program will be an affiliation for student nurses of the Honolulu hospitals, public health nurses, and nurses in other fields of nursing, and will be conducted at Leahi Hospital under the immediate direction of DR. H. H. WALKER.

It is expected that there will be an appreciable demand, in the postwar health programs, for nurses who have had special training in tuberculosis nursing. This program, which is being sponsored by the Tuberculosis Association, was suggested at a meeting of a group of nursing educators whose interest was to improve the education and standards of nursing in the territory.



# What's the other thing we ought to do this Christmas?

FOR the last four years, the Christmas phrase "Peace on earth, good will to man" has had a pretty hollow, bitter ring.

This year, it won't.

And surely, one thing each of us will want to do this Christmas is to give thanks that peace has finally come to us—both peace—and victory.

One other thing we ought to do:

In our giving, this year, let's choose—first—the kind of gift that helped to bring us peace and victory and will now help us to enjoy them.

★

Victory Bonds take care of the men who fought for us—provide money to heal them, to give them a fresh start in the country they saved.

Victory Bonds help to insure a sound, prosperous country for us all to live and work in.

Victory Bonds mean protection in emergencies—and extra cash for things we want to do ten years from now.

★

Choose—first—the finest gift in all the world, this Christmas.

Give Victory Bonds!

## Give the finest gift of all – VICTORY BONDS!